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CONNECTIVE TISSUE: A SYNTHESIS OF MODERN THOUGHT AND ITS IMPACT ON THE UNDERSTANDING OF NASAL DISEASE.*†‡

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The past 15 years have witnessed a dramatic change in the concept of body response to disease, primarily due to altered views about connective tissue physiology. The present paper is a review of the literature, correlated with clinical experience and substantiated by experimental work. A broad understanding of connective tissue can prepare otolaryngologists for the changing concepts of disease and therapy.

HISTORICAL BACKGROUND.

Virchow stressed the importance of connective tissue and the unity of all types of connective tissue. The following quotation from Theophile Bordeaux, a famous scientist of the Eighteenth Century, is an excellent description of the modern concept of connective tissue ground substance. "It is the most extensive and useful part of the body. It nourishes all the organs. It connects one with the other and favors

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and entertains their mutual relations. It is the seat of numerous diseases and the site of many manifestations of animal life."[1] Claude Bernard alluded to this in his discussion of the "milieu interne." During the Nineteenth Century, however, the study of cellular activity became more important. The concept of connective tissue slipped from a predominant position to that of a supporting structure lacking vitality and activity.

Interest in connective tissue grew with the industrial chemists' study of leathers, using the techniques of crystallography and X-ray diffraction. Progress was made when Duran-Reynals[2] discovered that testicular extract increased the activity of vaccinal virus. In 1933, Klinge[3] compared rheumatic fever lesions with those in animals receiving protein injections and postulated that the pathogenesis of rheumatic fever was due to mesenchymal ground substance alteration induced by allergy to bacteria.

The classical presentation of Klemperer, et al.,[4] defined diffuse collagen disease in 1949. He combined various diseases into a single group showing connective tissue alterations. Subsequently, the outburst of effort, primarily in the United States, Great Britain and the Scandinavian countries, clarified and filled in a few of the voids in our knowledge of the intercellular spaces.

This work is an attempt to organize the vast accumulation of fact and theory into a systematic study, to relate it to nasal problems and to provide a working hypothesis for further experimental study.

STRUCTURE OF CONNECTIVE TISSUE.

The connective tissue is a continuous matrix, the consistency of which varies from the loose jelly of the umbilical cord to the dense structure of bone. Within this matrix lie the interlacing fibrous structures, from which the tissue derives its name, and the cellular elements which appear to control its plasticity and metabolic activity. This matrix pervades all tissues. Its characteristics are determined by its location. Of primary significance is the fact that the connective tissue

is continuous throughout the body and that no organ system is free from the environment of the connective tissue.

I. Cells of the Connective Tissue.

A. Fibroblasts:

These originate from the primitive mesenchymal cells as do all the cells of the connective tissue. The morphological characteristics of these cells are described in standard textbooks of histology. The fibroblast controls the characteristics of the ground substance and responds to many stimuli in a rapid, active fashion.

The fibroblast is the most common cell of the connective tissue. It appears to have two important functions: the secretion of the precursor of fibers and of the acid mucopolysaccharides in the ground substance. Experimentally, the increase in response of the cell was found to parallel the increase in connective tissue ground substance.[5,6] Subsequent work has given further evidence of the importance of fibroblasts.[7-12]

The name fibroblast is really a misnomer. More properly it should be termed a mesenchymal cell, differentiated and undifferentiated.[13] The present study demonstrates the constant relationship between the undiffereniated mesenchymal cell and the ground substance.

B. Macrophages:

These are a second derivative of the primitive undifferentiated mesenchymal cell and are indistinguishable from the histiocytes; in fact, they are probably histiocytes which have developed phagocytic properties due to external stimuli.

C. Mast Cells:

These cells were given their name by Ehrlich in his first scientific publication in 1877. It has been suggested that the mast cells originate from mesenchymal cells in the adventitia of blood vessels.[14,15] The function of these cells is still open to question. These cells are particularly striking because of the metachromatic staining of the cytoplasmic granules which

differentiate them from the other connective tissue cells. Jorpes, et al.,[16] correlated the metachromasia with the Schiff positive granules. Since that time, there have been many studies on mast cells, especially by the Scandinavian school as represented by Asboe-Hansen.[17-19] Fawcett[20] showed that the mast cell granules were released as a functional part of the activity of the mast cell. Especially noteworthy was the fact that histamine liberators also caused extensive degranulation of the mast cells.

A number of functions have been attributed to the mast cells. Among these, are:

1. Defense. This has been related to the secretion of heparin by the mast cells. Experimental evidence showed their increased activity in areas irritated by carcinogens.[17,21-24]

2. Fat metabolism. Heparin is known to diminish lipemia. The mast cell relationship to atherosclerosis is still under investigation.

3. Hyaluronic acid secretion. It has been claimed that the mast cells are the primary source of this important ground substance factor. This is questionable and will be discussed later.[25-30]

4. Histamine production.[31,32]

5. Hormonal end organ. There is an increase in mast cells in myxedema and a decrease with thyroxin or in thyrotoxicosis. The controlling factor appeared to be the hormones of the pituitary which caused stimulation of the mast cells.[25,26] The adreno-corticoids caused a decrease in the number of mast cells with degranulation and vacuolation.[26-29,33]

II. Fibrillar Components of Connective Tissue.

A. Collagen:

This is the chief fibrillar component of connective tissue and consists of bundles of oriented fibers which stain intensely with picrofuchsin and the acid aniline dyes. Although collagen is histologically and chemically identical, it is found in different physical and functional states depending on its

location in the body. It ranges from the tough, heavy tendons and fascial sheath to the fine supporting lacework of the alveoli in the lung. Collagen is identified chemically by its chief amino acid, hydroxyproline, found only in this substance.[34]

The formation of collagen is still problematical. In 1947, Meyer[35] theorized that young fibroblasts secreted both hyaluronic acid and a soluble precursor of collagen into the ground substance. Acidification resulted in an insoluble fiber on the surface of which was the polysaccharide. As the collagen aged, the polysaccharide thinned and hyaluronic acid was gradually replaced by chondroitin sulfate.[36,37]

The electron microscope showed that the fibers have a very distinct periodic striation averaging 640 Angstroms and that collagenous fibers arose from the fibroblasts.[7,39] These fibers were embedded in an amorphous mucopolysaccharide ground substance.[38,39] Of particular interest was the discovery of procollagen by Russian workers and evidence of formation of collagen *in vitro*.[40]

B. Reticulin:

This substance is identified, as a rule, by silver impregnation. Its heaviest concentration is usually in the basement membrane of connective tissue where it forms an argyrophil network of fibers intimately associated with amorphous ground substance. Electron microscopy showed that the fibrils have the typical 640 Angstrom periodicity identifying collagen fibers. There was also some indication in this study that the reticulin fibers may change into collagen as they mature, forming a fibrous continuum.[41]

C. Elastin:

The exact nature of this fiber is unidentified. X-ray diffraction suggested a long chain of protein which appeared to be structurally similar to rubber and other elastic materials.[42,43] It was also intimately associated with the amorphous matrix of sulfated mucopolysaccharides and related to the collagens.

D. Oxytalan Fibers:

These have only recently been identified by Fullmer and Lillie.[44] Their exact significance is still obscure. They are located in areas where stress is distributed from one medium to another such as the junction of tendon and bone and at the periodontal membrane.

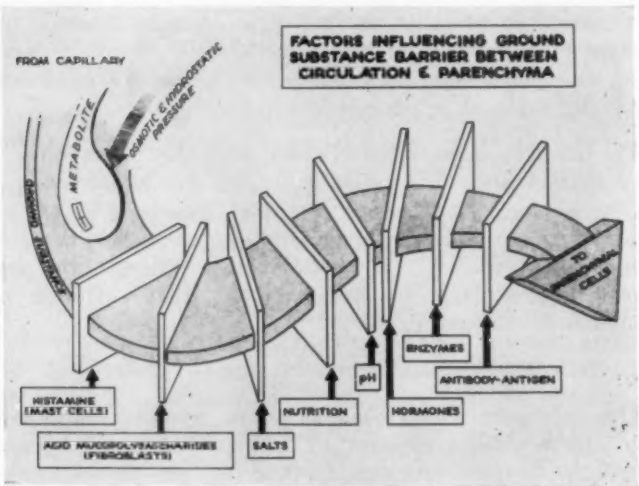


Chart 1. A diagramatic presentation of the role of ground substance on the functional state of the parenchymal cells.

III. Ground Substance in Connective Tissue.

This is the most important element of connective tissue activity and it is not demonstrated by ordinary histologic techniques. Its very presence was questioned until proven by Bensley[45] in 1934. The ground substance is the amorphous colloid in which lie the cells, fibers and vessels of connective tissue. It consists of serum-like components of tissue fluid, local cellular metabolites, hormones, minerals, etc., and the factors distinctive to the connective tissue—the acid mucopolysaccharides. The latter are the paramount factors in the functional activity of connective tissue.

A. Biochemical Studies:

The most intensive work has been done by Karl Mayer and his group.[46-54] The chemical substances forming the gel of the ground substance are the acid mucopolysaccharides which are high polymers of hexosamine. These are long chain flexible polymers with a very high negative charge and a strong affinity for cations and water molecules. Their physical molecular structure gives them a very high viscosity in low concentration. The physio-chemical changes found in the ground substance relate directly to the changes in state of polymerization and pH which govern the gel-sol state.

1. Hyaluronic acid. This is the most important mucopolysaccharide. It is found in practically all connective tissue. It is preeminent in the maintenance of the plastic, labile, functionally active ground substance. Hyaluronic acid has been found in various degrees of polymerization, its molecular weight ranging from 20,000 to over 8,000,000. The polymers form coiled chains which are hydrophilic and maintain water in physical union. When depolymerized, either by bacterial or testicular hyaluronidase (the spreading factor), the polymer linkage is broken and yields acetyl glucosamine and glucuronic acid. Hyaluronic acid is the only mucopolysaccharide to be hydrolyzed by bacterial hyaluronidase.

2. Chondroitin sulfates. Chondroitin sulfate A is the chief acid mucopolysaccharide of hyalin cartilage. Chondroitin sulfate B is identical with Chondroitin sulfate A except for minor physical properties. It is an important constituent of skin and other loose connective tissue and increases with age. It is associated with heavier formations of gross collagen and diminished tissue activity. It is characterized by resistance to any hyaluronidase activity. The other sulfated mucopolysaccharides are Chondroitin sulfate C, Chrondroitin and Kerato-sulfate.

B. Hyaluronidase Studies:

This is the most important and best studied of all of the spreading factors. This enzyme has the unique property of depolymerizing the acid mucopolysaccharides and significantly

altering their responses. This changes the gel to a sol, causing increased permeability and water solubility with resulting increase in the spread of particles in the tissue.

The chief commercial source of hyaluronidase is testicular extract from cattle. Other animal sources are snake and leach venom. Clinically, its spreading effect has been used in hypodermoclysis and local anesthesia. It depolymerizes all the acid mucopolysaccharides except Chondroitin sulfate B. Bacterial hyaluronidase is prepared from cultures of streptococci, pneumococci, staphylococci and Clostridium welchii. These enzymes will not depolymerize the sulfated mucopolysaccharides.

Other substances implicated as spreading factors are mammary gland extracts, thyroid, azoproteins, ascorbic acid and mercurial diuretics among others.[55] The nature of their action is still speculative.

C. Biological Studies:

The ground substance and its cells interact in a constant, highly dynamic equilibrium of limited stability. Interest in the ground substance was stimulated with the knowledge of the spreading factor. Duran-Reynals[2] and Meyer[35] first discussed the interrelationship of hyaluronidase and hyaluronic acid in connective tissue. In 1949, Gersh[56] and Catchpole[6] presented one of the early analyses of the connective tissue ground substance. They emphasized the physiological activity of the ground substance and response to injury. Angevine[57] stressed the importance of the hematoparenchymal barrier. A substance passing to the tissues must first travel through the capillary wall, through the connective tissue ground substance, and then through the cell membrane. Any change along this path altered the metabolism of the cell. The mechanism of fluid transport was demonstrated to be due to molecular capillarity along connective tissue fibrils.[58] Edema with increased hydrostatic pressure accentuated the spreading. This may explain the activity of many of the anti-spreading factors used clinically such as epinephrine, morphine, the anti-histamines, salicylates, rutin and adrenal-cortical extracts.

1. Inflammatory States. The response of the ground substance to inflammation is of great clinical interest since it leads to an understanding of the fundamental processes in disease. It was shown that injury caused immediate increase in capillary permeability with edema, accumulation of lactic acid and drop in pH, followed by an infiltration of blood and tissue cells.[59] The invasion of mesenchymal cells paralleled the increase in the acid mucopolysaccharides as shown by increased metachromasia.[60] The maximum amount of mucopolysaccharide was found in tissues characterized by glycolytic metabolism.[61] Injection of hyaluronidase into rat skin connective tissue resulted in the picture of early trauma. The ground substance showed evidence of depolymerization, metachromasia was lost, collagen fibers separated, the mast cells were disrupted and the fibroblasts swollen and vacuolated.[62] Hyaluronidase also caused increased permeability in semipermeable membranes.[63] Marked increase of tissue hyaluronidase was found after injury.[64] Other substances such as proteinases have been implicated in the depolymerization process, which controls the gel-sol state of the ground substance.[65] The rapid rate of turnover of hyaluronic acid was shown by S^{35} studies.[66-68]

The above experiments have demonstrated two factors involved in early inflammation: one was increased capillary permeability and edema due to histamine and hyaluronidase; the other was depolymerization and tissue disorganization caused by hyaluronidase and other factors. A "vicious cycle" was thus started. The edema acted to maintain and increase the spreading by hydrostatic pressure.[69]

Infection and its spread is particularly pertinent since a large proportion of the inflammation seen by rhinologists is due to infection by bacteria and viruses in the presence of hyaluronidase[2,70] Streptococci from throat infections and otitis media produced hyaluronidase quite consistently.[71] No correlation between the virulence of streptococci and the formation of hyaluronidase was found.[72-75] Some have even suggested that hyaluronidase has a protective effect.[72]

The importance of the hyaluronic acid-hyaluronidase system

in infection is substantiated by the presence of a non-specific enzyme "anti-invasin." This destroys hyaluronidase and is a part of the essential defense mechanism of the body to prevent invasion during severe infections.[76,77] Subsequent work has shown that there was an increase in the inhibitor in many unrelated infections.[78,79] The increased serum inhibitor appeared to be a response of the organism to hyaluronidase released by the tissues themselves.[80] The anti-hyaluronidase role of heparin was suggested by the concomitant increase of heparin and "anti-invasin" in the blood.[81,82]

It has been demonstrated that the increase in acid mucopolysaccharides is a step in the process of inflammation. This reaction is non-specific and was found in a wide variety of diseases including rheumatic fever, arthritis, diabetes, myxedema, pregnancy, allergic reactions, prolonged administration of estrogen, androgen or desoxycorticosterone, inflammatory lesions due to bacteria or viruses, cold, trauma, anoxia and radiation.[61] The degree of response depended upon tissue specificity. Some investigators suggest that the eventual stigmata of these diseases was due to deterioration of the acid mucopolysaccharide itself.[3,4,6,19,56,83-86] Others, particularly the German school, believed that the ground substance was not important in the formation of fibrinoid but that it was due to the influx of extrinsic material from fibrin or serum protein.[87-89]

2. *Hormonal Effect on the Ground Substance.* Response of connective tissue to hormones has been observed clinically for many years. Rhinologists are aware of the tissue changes associated with the menstrual cycle and pregnancy.[90,91]

a. Adreno-corticoids. The theory of the General Adaptation Syndrome by Selye[92] in 1946, aroused general interest in the adrenal corticoids. Cortisone and ACTH depressed the activity of all connective tissue elements.[28,93-97] The decrease in acid mucopolysaccharides as demonstrated by the absence of metachromasia has been considered a prime factor in delaying fibroplasia and healing.[98] Lack of hyaluronic acid diminished the tensile strength of wounds.[99] Its addition to wounds inhibited by Cortisone stimulated fibroplasia.[100] Cor-

ticoids also diminished the activity of the mast cells.[34,101] Atrophy of epidermal structures has been reported[102] showing the close functional relationship of connective tissue to neighboring structures. Cortisone acts at the level of the cell membrane and may be an "antihyaluronidase" factor. The permeability of semi-permeable membranes was decreased by the corticoids.[63] This effect was seen on capillary permeability[103] and was associated with vasoconstrictive factors on an autonomic basis.[104,105] It caused decreased invasiveness of experimentally produced carcinomata.[106]

Desoxycorticosterone caused a reversal in response. The granulation tissue was exuberant with young giant fibroblasts (mesenchymal cells) showing great activity and with a heavy ground work of collagen. Similar results were seen with testosterone.[107] Anterior pituitary somatotropic hormone also prevented the inhibitory effect of Cortisone on granulation tissue[108] and the massive systemic infection of multiple abscesses seen after giving Cortisone and ACTH to experimental animals[109] Hypophysectomy decreased the production of hyaluronic acid and chondroitin sulfate.[110] Anterior pituitary growth hormone increased this acid mucopolysaccharide production.[110,111] The adrenal-pituitary axis appears to be an important and delicately balanced mechanism in the activity of the connective tissue.

b. Thyroid hormone. Articles in the past have alluded to the role hypothyroidism plays in vasomotor rhinitis.[112-114] The use of thyroid hormone in otolaryngological conditions has been questioned. Walsh,[115] in 1950, advocated the use of thyroid hormone as determined by history and clinical findings. Further evidence relating the hypometabolic state to thyroid hormone has been presented on a theoretical basis.[116] Increase in hyaluronic acid and chondroitin sulfate was found in pretibial myxedema[117] and in hypothyroid myxedema.[118] This has been explained by the action of the thyrotrophic hormone which stimulated acid mucopolysaccharide production.[119,120]

c. Sex hormones. The relationship between sex hormones and the nose has received clinical attention.[90,91,121] The changes

in the sex skin of the Macaco monkey are related to the menstrual cycle. This was shown to be controlled by estrogens.[30,122] The material causing the edema was hyaluronic acid and hyaluronidase caused immediate softening[123] Testosterone presented an identical picture in the cock's comb[124] and has been related to gonadotropic hormones.[125] Gonadotropin decreased polymerization with increased tissue permeability and spread of tuberculosis in rabbits.[126] The gonadotropic-sex hormone mechanism probably involves the adrenal cortex as well.[127]

Relaxin, a sex hormone associated with pregnancy, has a profound effect on the connective tissue. It has a specific action on the connective tissue of the symphysis pubis, loosening the joint in preparation for delivery.[128] On theoretical grounds this evidence for specificity of connective tissue response could explain the changes in the nasal and laryngeal tissues due to hormonal factors.

d. Parathyroid hormones. The parathyroid glands have been involved in connective tissue ground substance. Parathyroid extract depolymerized the ground substance. Cases of fibrous dysplasia showed a high serum mucoprotein and suggested the interrelationship of the parathyroid and the ground substance.[129,130] Evidence permits only speculation as to the possible etiology of otosclerosis, a connective tissue disease. The localization of the otosclerotic focus may be a specific connective tissue response in the embryonal cartilage of the fissula ante-fenestrum.

3. Ascorbic Acid. Ascorbic acid is the primary vitamin implicated with connective tissue functions as shown by the clinical course of scurvy with increased capillary fragility, petechiae and poor wound healing. The ground substance was depolymerized as evidenced by a rise in serum glycoprotein. Normal collagen was not formed and the existing collagen was weakened due to its abnormal matrix.[131-136] Ascorbic acid is an essential factor in connective tissue formation as shown by its chemical relationship to hyaluronic acid[137] and the similarity between scorbutic tissue and that of collagen diseases.[138]

HISTOCHEMICAL TECHNIQUES.

The combined efforts of biochemists, biophysicists, anatomists, histologists and the clinicians have developed many ways to study connective tissue. All of these methods have very distinct limitations which have been constantly re-emphasized by workers in the field.[46,139] The present study has stressed histochemical methods[140-153] as the most useful for clinical study.

Histochemical technique differs from ordinary histologic technique in that the structural organization of the tissue is correlated with its functional state. The limitations of histochemical studies have frequently resulted in the completely contrary findings reported in the literature. A number of factors may explain this. One of the most important is that the presence of many constituents in the tissue, such as protein, may obscure the stain.[154-156] Some of the investigations on histochemical stains were done on chemically pure substances and in many instances, the results differed from those found in tissue[150,157] since chemically pure substances are never found in life. When the stains were used on the tissue complexes, they were effective.[158,159]

I. Stains.

Three stains have been most widely used because of their specificity, sensitivity and reproducibility.[11]

A. Periodic Acid Schiff (PAS):

This stain [140,141] was considered to be the least specific in this study. It stains the acid mucopolysaccharides and also stains glycogen, mucoproteins, glycoproteins, collagen, gelatin, reticulin, plasma and serum.[101] The PAS material stains a vivid pink to red. Using the controlled simultaneous preparation method,[11] no difference was found between the staining characteristics of tissue, prepared serially, with and without hyaluronidase. The experiment showed that factors other than acid mucopolysaccharides were stained; therefore, the PAS stain was not used in the present study.

B. Metachromatic Reactions:

The most common dyes used are the basic dyes such as thionin, azure A or toluidine blue. Ehrlich used the term "metachromasia" to indicate that certain tissues and structures stain in a different color than that of the original dye solution. The alteration is based upon electro-chemical changes in the dye molecule.[160] The metachromatic reaction is an important source of information about the acid mucopolysaccharides of the ground substance. They will stain metachromatically, depending upon two factors: the state of polymerization and the amount of sulfation. The toluidine blue method was used in this study.

C. Colloidal Iron Adsorption:

This staining technique was first reported by Hale[142] in 1946 and has been modified[145] Acid mucopolysaccharides will adsorb colloidal iron which then stains positively with the Prussian blue reaction of ferrocyanide. The collagen is stained with picro-fuchsin. A modified Rinehart and Abul-Haj stain[145] was used in this study.

EXPERIMENTAL DATA.

This work originally began as a study of nasal tissue in a variety of vasomotor states. An attempt was made to correlate vasomotor rhinitis with various conditions involving the endocrines such as pregnancy, hypothyroidism and changes of the menstrual cycle. It was discovered that the routine histologic stains presented little information about the functional state. The development of histochemical stains provided the means for visualization of these changes.

I. Normal Turbinate Mucosa.

A series of biopsies were taken from normal middle turbinates. No vasoconstricting agents were used. The normal tissues were used to develop a standard technique and to study the variations in the range of normal connective tissue, Weisskopf and Burn.[11]

Metachromasia was demonstrated with toluidine blue. The

most striking violet color was in the intima of the arterioles and the connective tissue immediately around these vessels. Associated with the vessels were the very prominent mast cells. They were large, round or oval cells with the nucleus almost completely obscured by the concentration of deep violet granules. Occasional mast cells were seen with granules scattered into the surrounding medium, suggesting an actual secretion by these cells rather than mere mecnanical disruption. An occasional mast cell was also seen in the lamina propria among the mesenchymal cells, fibroblasts, and other cellular components. The average fibroblast is small and dense with minimal cytoplasm visible. Its nucleus is pyknotic without the prominent nucleolus seen in the active fibroblast. The ground substance of the lamina propria did not stain nor did the basement membrane. It appeared to have a perfectly clear background except for the faint suggestion of fine blue fibrils. The mucous glands varied in staining reaction from the orthochromatic blue to a very vivid metachromatic violet. Of interest was the metachromasia seen in the ciliated epithelium, mainly concentrated near the ciliated border, indicating their functional activity in the production of mucus. Incubation with testicular hyaluronidase (Wydase®) * substantiated the experimental evidence. There was loss of metachromasia in and about the blood vessels but no change in the mast cells or the mucus producing cells. With bacterial hyaluronidase, there was a persistence of faint metachromasia about the vessels. This suggests that both hyaluronic acid and chondroitin sulfate B were present. The heparin in the mast cells remained unchanged as did the neutral mucopolysaccharides of the mucus.

The colloidal iron stain corroborated and enriched the picture derived from the toluidine blue stain. Again the greatest concentration of acid mucopolysaccharides was found in and among the blood vessels. It was seen to form an important part of the structure of the vessel wall. The fibrillar material, now determined to be collagenous by its brilliant red stain, was surrounded by the fine blue mucopolysaccharide layer which pervaded everywhere. The clear ground substance

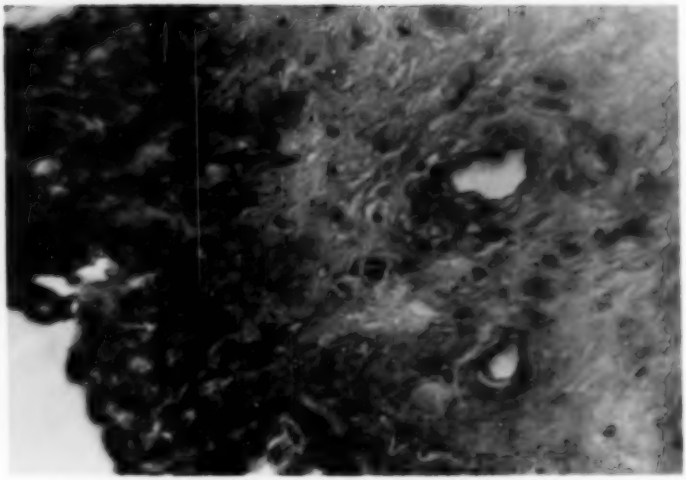
*Wydase® was kindly supplied by Wyeth Laboratories, Inc.

seen with toluidine blue was found to contain fine collagen granules and the acid mucopolysaccharides. The colloidal iron stain distinguished the unsulfated and more depolymerized acid mucopolysaccharides as compared to the toluidine blue. The basement membrane was beautifully demonstrated and given some functional significance. It was approximately seven to 15 micra thick and was seen to consist of fine amorphous blue staining ground substance intermingling with the red fibrils of reticulin. There was a continuity between these fibrils and the heavier collagen of the lamina propria. The greatest density of ground substance was found in the peripheral half of the basement membrane, gradually becoming less blue as it streamed into the lamina propria. The mucus likewise stained blue. Hyaluronidase incubation was again enlightening. There was a marked "washing out" of the background, revealing fine, red interlacing fibrils and fibers of collagen. The reticulin network of the basement membrane was more evident. There was persistence of some fine blue staining in the basement membrane as well as in the immediate vicinity of the vessels.

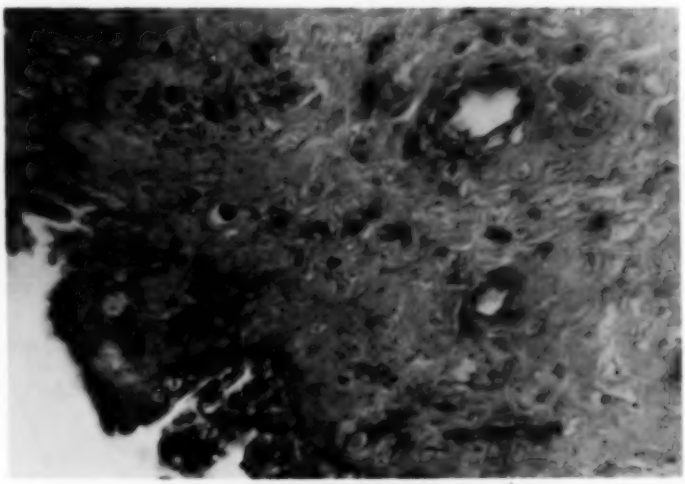
Certain facts may be assumed about the normal state of the connective tissue in the nasal turbinates. The basement membrane shows little depolymerization with bacterial hyaluronidase and therefore must consist chiefly of the chondroitin complex. The lamina propria contains acid mucopolysaccharides which appear to be chiefly hyaluronic acid with chondroitin sulfate B in a smaller amount. They appear to be in a relatively unpolymerized state as shown by lack of metachromasia. Increased polymerization and activity is found about the blood vessels. The presence of a relatively greater amount of hyaluronic acid as compared to chondroitin sulfate B suggests again the actively labile well hydrated nature of the turbinate mucosa.

II. Allergy and Nasal Polyps.

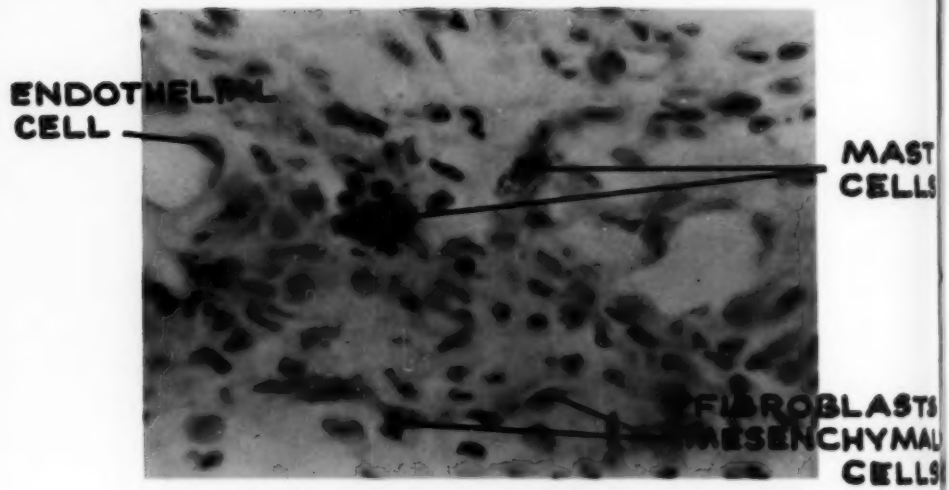
Once the consistency of these histochemical techniques was determined, the study was extended to various disease states. The relation of allergy to mesenchyme has been postulated on theoretical grounds.[161,162] Evidence suggesting nasal connec-



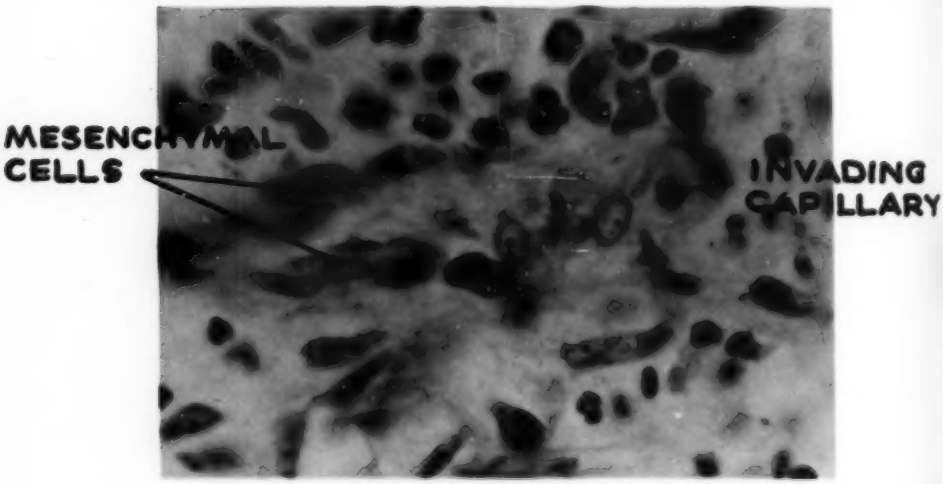
1. Normal middle turbinate. 430x. Colloidal Iron. The ground substance of the basement membrane and lamina propria are stained blue. The collagen fibers stain red.



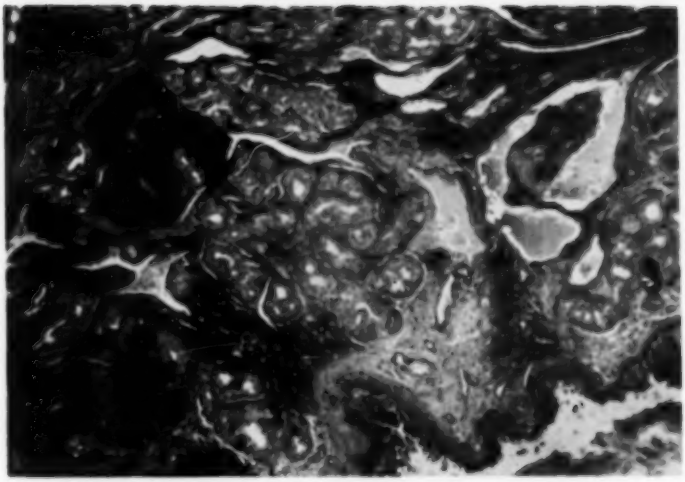
2. Normal middle turbinate. 430x. Colloidal Iron after Hyaluronidase incubation. The loss of blue stain confirms the presence of Hyaluronic acid. The persistence of a fine blue residue in the ground substance and blood vessel wall suggests the presence of Chondroitin Sulfate B.



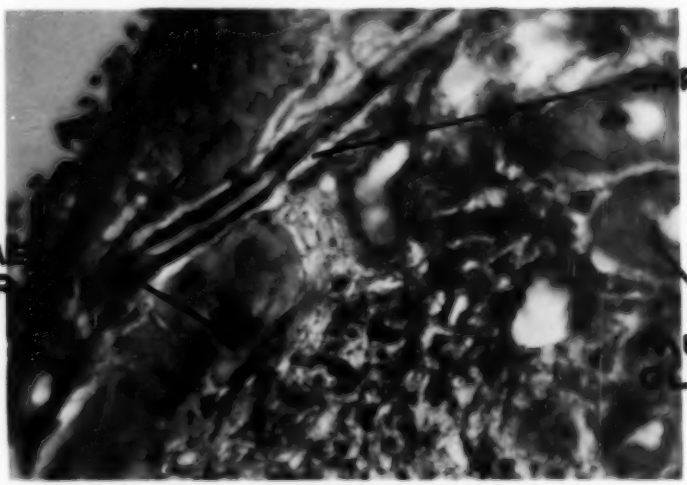
3. Normal middle turbinate 970x. Toluidine blue. The metachromatic granules of the mast cells are striking. There is a faint metachromasia in and about the capillary walls.



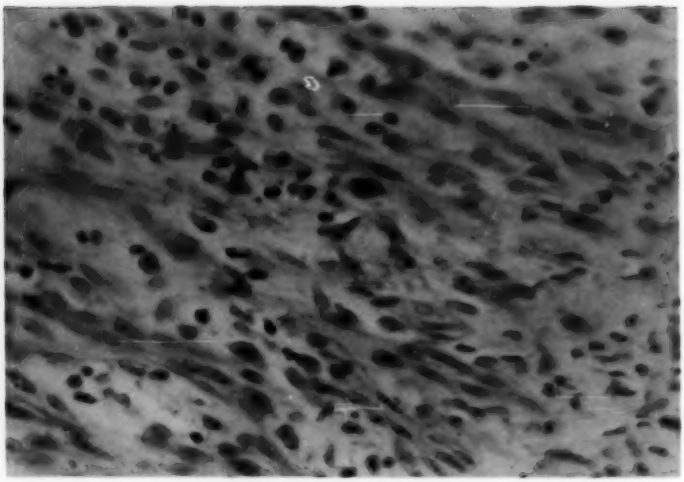
4. Laryngeal granuloma 970x. Toluidine blue. The large fibroblasts (mesenchymal cells) and the surrounding metachromatic ground substance reveal the active nature of this granulation tissue as compared to the normal Fig. 3.



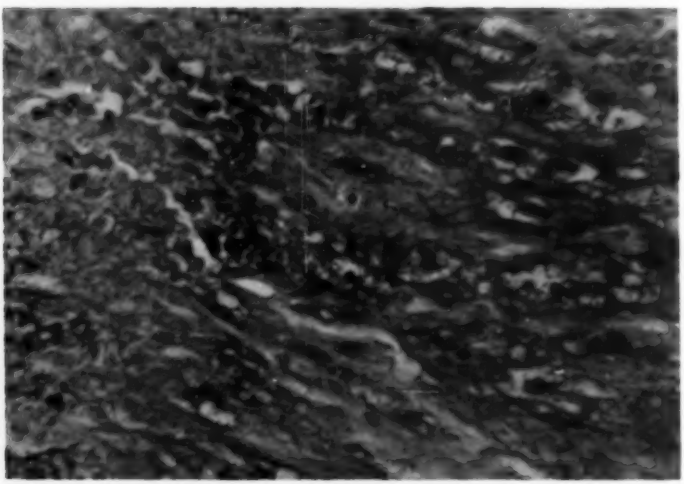
5. Middle turbinate, hypothyroid. 100x. Colloidal Iron. Note increased mucous glands and the density of the blue staining ground substance.



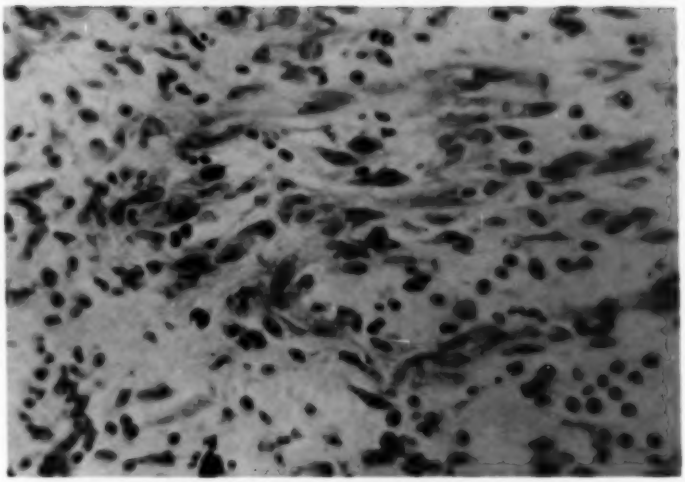
6. Middle turbinate, hypothyroid. 430x. Colloidal Iron. The increased ground substance of the basement membrane and lamina propria is striking.



7. Laryngeal granuloma 430x. Hematoxylin and eosin. The invading capillaries and mesenchymal cells are seen but no evidence of ground substance activity.



8. Laryngeal granuloma 430x. Colloidal Iron. Compare with Fig. 7. The streamers of fibroblasts are embedded in the acid-mucopolysaccharide.



9. Laryngeal granuloma. 430x. Toluidine blue. The metachromasia about the mesenchymal cells correlates well with the blue stain of Fig. 8.

tive tissue breakdown and depolymerization in allergy was presented in 1953.[163] Further evidence was presented[164] that the ground substance was involved in allergy and in the formation of nasal polyps. Study of nasal polyps gives considerable insight into the formation of the polyp and the functional mechanisms in the connective tissue. The polyp was found to contain acid mucopolysaccharides in varying states and concentrations even in the so-called edema fluid. Areas of increased activity in polyps were characterized by "puddles" of acid mucopolysaccharides showing metachomasia. Almost invariably, this was associated with the large active mesenchymal fibroblasts which are assumed to be the source of the mucopolysaccharide. Mast cells did not appear to play an important role and were conspicuous by their rarity. On the other hand, this might indicate their extreme activity with the absence of granules making them unrecognizable. There was relatively little collagenous material as compared to the normal nasal mucous membrane. The basement membrane was atrophic, absent or frequently replaced by collagenous fibers with relatively little ground substance. The picture suggests activity of the connective tissue with formation of an abnormal ground substance. This does not form the normal collagen associated with inflammatory changes. Further work is contemplated, involving ascorbic acid and allergies.

III. Infection.

A difference was found between acute and chronic infection suggesting the steps leading to collagenous healing. The acutely infected membrane showed the extensive infiltration of acute inflammatory cells, edema and areas of granulation tissue showing new capillary formation. The large accumulation of mucopolysaccharides was impressive in the areas of capillary formation and among the concentrations of the large mesenchymal cells. These, characteristically, showed foamy and granular cytoplasm and a large nucleus and nucleolus.

The chronic infection showed the presence of increased ground substance mucopolysaccharides, interspersed with dense collagenous fibers. The total amount of ground sub-

stance was less than in the acute stages. It presented a vivid picture of the outcome of chronic infection with collagen formation scarring and decreased vascularity. The decreased ground substance activity results in the eventual inability of the tissues to respond to the stress of infection.

IV. Pregnancy.

The most striking feature in the vasomotor rhinitis of pregnancy was the bulk of mucous glands which appeared to fill practically the entire section. This was associated with numerous large cavernous areas. With toluidine blue, the slides appeared to be "washed-out" having practically no areas of metachromasia even in the blood vessels. They appeared more like normal tissues which had been subjected to hyaluronidase. The colloidal iron stains substantiated this impression. There was a definite decrease in the amount of ground substance. The basement membrane was either atrophic or non-existent. All the cases were from the fourth to the seventh month of pregnancy, a period associated with increased estrogens. To understand these changes, the gonadotrophic hormone levels must be considered.

V. Thyroid Dysfunctions.

It was difficult to obtain turbinate specimens in clear-cut cases of glandular dysfunction which had not already been treated. Four cases were studied: two hypothyroid (one with true myxedema), one thyrotoxicosis and one case with complete ablation of pituitary activity by Synchrotron irradiation. The most persistent finding in all cases was the hypertrophy of the mucous glands. The hypothyroid states, including the dyspituitary, showed a marked increase in ground substance with the colloidal iron stain.

Turbinate tissue from the patient with pituitary ablation showed the most striking "puddling" of amorphous material, obliterating the normal lamina propria in many areas. There was almost complete absence of the normal cellular infiltration. The majority of cells present were the large mesenchymal cells associated with formation of ground substance. Even the normally flat endothelial cells of the capillaries

showed large nuclei with prominent nucleoli very similar to those of the mesenchymal cells. Since theoretically, no thyrotropic hormone is secreted after pituitary irradiation, this suggests the relationship of other hormones to ground substance activity. The increase in ground substance of the other two hypothyroid cases was not as dramatic, but was distinctly more than normal. The thyrotoxicosis case showed less than normal ground substance as evidenced by the visibility of the collagenous material in the lamina propria and the basement membrane.

The increased turgidity of the turbinates in the hypometabiloc states was reflected in an increase in amount and polymerization of the ground substance. Associated with this was a mucous gland hypertrophy, explaining the excessive nasal secretions. Both pregnancy and hypometabolic states are associated with hyperactivity and hyperdevelopment of mucous glands. The relationship of the mucous glands to these various states deserves further study.

VI. Atrophic Rhinitis.

The most prominent change in the three cases was the metaplasia into stratified squamous epithelium. The absence of mucous glands, the arteritis, and the collagen deposits in the walls of the arterioles were striking. There was an increase in the amount of mucopolysaccharide of the ground substance associated with an increased amount of collagen. This was paralleled by a marked increase of mesenchymal cells of the large primitive type and increased numbers of small capillaries. There was no lack of vascularity to explain this lesion. Of particular interest was the presence of a large number of mast cells. In selected areas, the picture of a blood vessel associated with collagen, active fibroblasts and mast cells was reminiscent of juvenile angiofibroma.[165]

Treatment with corticoids caused a remission in clinical symptoms and showed a dramatic change in the turbinate. There was diminution in all elements of the connective tissue. The ground substance appeared less dense. There were fewer mesenchymal cells present and those remaining appeared

smaller and more typically mature. There was diminution in mast cells as well as in the density of inflammatory cell concentration. At least part of the therapeutic effect of the corticoids[166-168] can be seen to be due to their inhibiting action upon the connective tissue components and corroborates the experimental work cited in the literature.

VII. Granuloma.

Lastly, a case of laryngeal granuloma was studied to compare a naturally occurring granulation tissue with those experimentally produced and reported in the literature. The findings were essentially those described by other workers[9,60,84,93,98,99] and helped corroborate the authenticity of our own histochemical stains and interpretations. The laryngeal granuloma was quite typical histologically. Histochemically, the greatest amount of ground substance appeared in the greatest area of growth activity. This was the junction between the amorphous fibrinous debris and the invading capillaries and mesenchymal cells. The same typical, large fibroblasts were seen and again appeared to be related to the formation of the acid mucopolysaccharides. There was practically no evidence of mast cells in significant numbers.

DISCUSSION.

Evidence has been presented that connective tissue is an anatomical, functional and biochemical unit. Since the connective tissue matrix is all pervasive, it must eventually be involved in all body processes.

Attention has been directed to the characteristic response of connective tissue in a variety of diseases and functional states. This response consists, primarily, of a series of physico-chemical changes in the acid mucopolysaccharides of the ground substance and is related to the activity of the mesenchymal cell. This intricate process is outlined in a hypothetical diagram in the accompanying chart (see Chart 2).

Experimental data is presented which suggests, at least within the limits of a preliminary survey, that many of the pathologic states of the nasal turbinates are reflected in

connective tissue changes. Vasomotor rhinitis, to name one, is more clearly understood in the light of these changes. This was predicted on theoretical grounds.[11] The close interrelationship of the nasal tissues to other body functions is also demonstrated on the histologic level.

It is felt that these studies may have clinical application. There is evidence that a variety of materials affects the

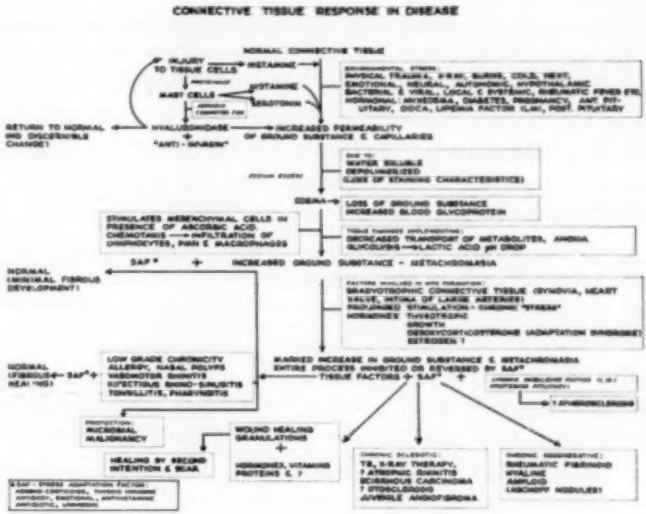

CONNECTIVE TISSUE RESPONSE IN DISEASE
NORMAL CONNECTIVE TISSUE
INJURY TO TISSUE CELLS
HISTAMINE
MAST CELLS
HEPARIN
HYALURONIDASE
"ANTI-SPREAD"
INCREASED PERMEABILITY OF GROUND SUBSTANCE & CAPILLARIES
WATER SOLUBLE (DEPOLYMERIZED) LOSS OF STAINING CHARACTERISTICS)
EDEMA
LOSS OF GROUND SUBSTANCE INCREASED BLOOD GLYCOPROTEIN
INCREASED GROUND SUBSTANCE = METACHROMASIA
NORMAL (MINIMAL FIBROUS DEVELOPMENT)
MARKED INCREASE IN GROUND SUBSTANCE & METACHROMASIA
ENTIRE PROCESS INHIBITED OR REVERSED BY SAP
TISSUE FACTORS
LOW GRADE CHRONICITY ALLERGY, NASAL POLYPS VASOMOTOR RHINITIS SUPERFICIAL RHINO-SINUSITIS TONSILLITIS, PHARYNGITIS
PROTECTION: MICROBIAL MALIGNANCY
WOUND HEALING GRANULATIONS
HEALING BY SECOND INTENTION & SCAR
HORMONES, VITAMINS PROTEINS ?, ?
?, X-RAY THERAPY, ? ATROPHIC RHINITIS SCIRRHOUS CARCINOMA ? CYTOCLASIS ? JUVENILE ANGIOFIBROMA
RHEUMATIC FIBRINOID HYALINE AMYLOID LIPOCHROMY NODULES?

Chart 2.

ground substance in general. These range from the chemical to the hormones and vitamins. It would be expected that some of these should exert a similar effect upon the nasal ground substance and may provide suggestions for the utilization of new therapeutic agents. The histochemical picture presents evidence of functional changes in the tissues and may be used to study the pharmacological actions of the agents. This was done in one case of atrophic rhinitis treated with prednisolone.

The ear, nose and throat are readily studied since specimens may be obtained in a functional state at surgery and from biopsies.[11,163,168,169] This contrasts with other investigations which have used animal experiments or post mortem material.

SUMMARY AND CONCLUSIONS.

1. A review of the literature presents evidence that connective tissue behaves as an anatomical and functional unit.

2. The ground substance is the most important component. It responds characteristically to a wide variety of conditions. The degree and site of response depends upon tissue specificity.

3. The mesenchymal cell is considered to be the ultimate source of the acid mucopolysaccharides, the active factor in the ground substance.

4. Histochemical investigation of turbinate biopsies in a number of disease states reveal connective tissue changes that correlate well with the experimental findings of investigators in other fields.

5. The connective tissue changes observed add to a further understanding of the processes involved in the vasomotor rhinitis of allergy, pregnancy and endocrine dysfunction.

6. Histochemical methods were suggested to evaluate therapeutic agents. One case of atrophic rhinitis, treated with prednisolone, was presented.

7. A new approach to the study of such connective tissue diseases as laryngeal nodules and otosclerosis is suggested.

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THE FRACTURED MANDIBLE.

A Method of Treatment for the Otolaryngologist.*†‡

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INTRODUCTION.

Today, because of fast cars and super highways, many disastrous accidents occur in rural areas, and the small community hospital becomes responsible for the total care of the critically injured patient. The attending otolaryngologist may be among the first called to treat the patient with injuries of the face, nose or even the mandible. Although the fractured mandible is usually reserved for the oral or maxillofacial surgeon, these specialized services may not be readily available, and under these circumstances the otolaryngologist must have a basic knowledge of the problem and a satisfactory method of treatment. Since most otolaryngologists have no experience with these cases during their residency training, they are unfamiliar with the principles of good treatment and often do not know where to begin.

Our objective has been to study this subject and from the abundant information in the literature and from our personal experience distill a method of treatment of the fractured mandible which can be applied by the otolaryngologist trained in head and neck surgery. I believe that the otolaryngologist, using easily available equipment, working with the local dentist, can satisfactorily treat the majority of fractured mandibles.

SURGICAL ANATOMY.

The surgical anatomy of the mandible can be divided into

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the mandible proper, with its nerves, blood vessels and muscles; and the teeth.

The Mandible Proper.

The mandible consists of a condyle, neck, coronoid process, ramus, angle, body and symphysis.[1] The bony structure varies in the different areas, and this is clinically significant. The condyle or head is broad, and it is separated from the mandibular fossa of the temporal bone by the cartilaginous meniscus and is surrounded by a fibrous capsule. Fractures within the capsule involving the head of the condyle are rare.[2] The neck is slender and when under stress will fracture preventing injury to the middle cranial fossa.[3] The external pterygoid muscle tendon inserts into the anterior margin of the capsule and partially into the neck. The coronoid process develops from the pull of the temporal muscle similar to the development of the mastoid bone from the pull of the sternocleidomastoid muscle, and it is seldom fractured. Two plates of compact bone, separated by a core of cancellous bone, make up the ramus. It is covered on its lateral surface with the masseter muscle and medially by the internal pterygoid. Because of this double muscular covering, the ramus when fractured seldom becomes displaced. As the ramus broadens into the thicker body there is a line of structural weakness along which fractures occur.[3] The body or horizontal ramus which lies anterior to the angle is thicker than the descending ramus. The two halves of the mandible are united in the midline, but a blow to the chin may separate this symphysis. The tooth bearing or alveolar portion is weaker than the main section of the body, and it may be fractured alone, without a fracture through the body.

There are two openings in the bone: the mandibular foramen, which is the entrance for the inferior alveolar nerve and artery, and the mental foramen which is the exit of the mental nerve, a cutaneous terminal branch of the inferior alveolar nerve. Except for an anesthesia of the lower lip, injuries to the mental nerve have little clinical significance. Within the ramus and body of the mandible lies the mandibular canal, through which passes the i[illegible] alveolar nerve

and artery. The nerve innervates the teeth and the mandible. The artery nourishes the bone and the teeth, and with the periosteal collateral circulation the mandible is quite vascular. Fragments of bone which have an attached periosteum should be preserved since the circulation may be adequate to maintain viability; however, if the blood supply to a tooth is disturbed, the pulp will die, and it is usually best that such teeth be removed before infection develops.

The muscles have a double significance. Their normal action provides extensive motility to the bone and when the mandible is fractured, they are important in the displacement of the fragments. The internal and external pterygoid muscles produce a side to side or chewing motion of the jaw. When a fracture occurs anterior to their insertion, the posterior fragment will be pulled inward and upward. The masseter and temporal muscles participate in the upper displacement of the posterior fragment. These four muscles of mastication or the posterior group, interact to produce a general pull which is upward and toward the midline. The anterior group, or the accessory muscles of mastication, consisting of the genio-hyoid, mylohyoid, digastric and genioglossus muscle, have a combined tendency to pull the anterior fragment downward and inward.

Because of the action of the muscles on the fragment, the fracture line can be classified as favorable or unfavorable.[5] In the unfavorable fracture, the fragments are kept separated by the muscular tension. When favorable, the fragments are aided in apposition by the muscles. The fracture line can be in a horizontal or vertical plane. The favorable type of fracture is partially immobilized by the muscle action and requires less surgical fixation to produce rigidity.

The Teeth.

The teeth are important surgically for several reasons. Their presence in the jaw, and this is particularly true of the third molar and the cuspid in the adult, furnish a potential line of weakness. In the child, the presence of unerupted permanent teeth weakens the structure of the mandible[3] but

this appears to be compensated by the increased elasticity of the bone.

The upper and lower dental arches articulate with the opposing facets of the teeth. The dental occlusion must be considered in the proper reduction of the fracture. The normal articulation of the teeth serves as a guide in the alignment of the fragments, and fixation is made restoring the patient's normal bite. If this is neglected in the process of reduction, the patient will become a dental cripple requiring considerable rehabilitation in the future. The surgeon should familiarize himself with the normal variations in dental occlusion and be aware of the fact that a patient's normal bite may be an abnormal one, such as a crossbite.

The teeth also serve as pegs for interdental and archbar wiring. There is a difference in the solidity of the deciduous and permanent teeth, and the primary teeth do not withstand stresses as well as the permanent teeth and may loosen or shift in position. This presents a special problem of handling a fracture in a child. When possible the permanent teeth should be used for immobilization and these erupt at different ages.

The edentulous patient, because of the lack of the precise articulating surfaces of the teeth, presents a different situation. Although some degree of variation in alignment may be compensated for by new dentures, the normal distance between the upper and lower dental arches should be maintained during the reduction of the fracture. This will prevent strain upon the temporo-mandibular joint and avoid future pain in this area.

CLASSIFICATION OF FRACTURES.

The general classification of fractures can be applied. The fracture may be *simple*, a linear crack with no external communication and usually seen between the condylar head and angle. It may be *compound*, either through the tooth bearing area or with a facial laceration. The *comminuted* fracture is the result of a violent force and frequently is also compound.

The *complicated* fracture usually involves the inferior dental nerve and vessels. The impacted fracture is rare, and there may not be any noticeable clinical movement. The surgical neck of the condyle may present a *greenstick* fracture. When a fracture occurs with minimal trauma one must be suspicious of a generalized or local skeletal disease causing this *pathological* fracture.

A special description of the mandible fracture can be based on the anatomical site of the injury:

1. Condyle.
2. Neck.
3. Coronoid process.
4. Ramus.
5. Angle.
6. Body.
7. Symphysis.
8. Alveolar process.

The fracture may be:

1. Single unilateral.
2. Double unilateral.
3. Bilateral.
4. Multiple.

Kazanjian has devised a useful classification based on the presence of teeth in the fragments.[4]

1. Class I—teeth are present on either side of the fracture.
2. Class II—teeth are present on one side of the fracture.
3. Class III—no teeth are present on either side of the fracture.

Classifying the fracture will crystallize the salient features of the injury and will help in determining the proper therapy.

THE HEALING PROCESS.

When the mandible is fractured, bleeding occurs between the ends of the fragments. The blood irrigates the wound and then clots, protecting the injured ends. Within 48 hours

new tissue starts forming from the mitosis of the fibroblasts and endothelium, and these cells invade the blood clot. By the third day the fibroblasts have produced collagen fibers, and capillary sprouts can be identified microscopically.[5] As part of the reparative process, phagocytes enter the injured area soon after trauma occurs and begin to remove the cellular debris, blood pigments and fibrin. The proliferation of the fibroblasts and capillaries continues, and a highly cellular granulation tissue results. After the first week, this tissue starts to immobilize the bony fragments; however, they can still be moved with pressure and any necessary correction in alignment can be made.[6] The fibroblasts develop a basophilic ground substance which surrounds the cells and as this callus is formed, the union between the fragments becomes firmer and greater forces are needed for realignment. The healing process continues with the absorption of bone by the osteoclasts and replacement by osteoblasts. Trabeculae are formed, shaped by muscle stresses until the fully differentiated bone is produced. The total process may take a year, but the mandible is rigid enough for function in about six weeks.

When comminution of bone is present, the fragments are united by this process and are incorporated in the final structure. A bone graft which replaces destroyed tissue, is invaded by the fibroblasts and in time becomes an integral part.

The lacerated epithelium, either the skin or oral mucous membrane, heals by proliferation from the epithelial edge. Injured muscle is replaced with fibrous tissue and if enough muscle remains it continues to function normally. The damaged blood vessels are replaced by collateral circulation. The severed nerves undergo degeneration of the distal end, but if carefully anastomosed, there may be some return of function although it may take over a year.

Rapid and uncomplicated healing of the fracture depends on several factors: *1.* the fragment ends must be in close apposition. The further the fragments are separated, the longer it will take to fill in the defect and a large defect may not be united by bone but by fibrous tissue, producing an unstable union. *2.* The fragments must be immobilized as

completely as possible. A minute amount of motion at the fracture line may stimulate proliferation of bone[2] but any additional motion will either cause shearing of the granulation tissue and injury to the circulation across the healing zone or it will produce unwanted tensions at the healing line which prevent the laying down of bone. A third factor is the presence of infection in the wound. It may reach the fracture site through several channels. Fractures through the tooth bearing area are frequently compounded into the mouth, and the fracture will be influenced by the amount of infection present there. Bacteria can be brought in through a skin laceration or by infected teeth which are adjacent to the fracture. The blood stream and lymphatics have a lesser role in infecting the area. Suppuration produces necrosis of the soft tissue and bone, preventing proliferation and bridging of new bone. In this environment, the healing process is slowed down and the dead bone or sequestrum must be removed before healing can continue. Foreign bodies also will act as sources for reinfection and will delay healing until removed.

When there has been a large loss of tissue, either soft or bony, infection is usually present. This type of injury heals by secondary intention. Granulations develop in the base of the wound and gradually will fill in the defect. Healing may be complicated by an osteomyelitis of the bone and the final union in this type of injury frequently is fibrous and unstable functionally. The solution is the elimination of infection with the removal of the sequestrum, and when this has been accomplished, the fibrous union must be replaced by a bone graft for stability.

The patient's nutrition is important for healing. Vitamin C is essential in the production of collagen and vitamin D is needed for the completion of calcification. Bone cells which are actively proliferating require generous supplies of oxygen, organic calcium salts, amino acids and proteins. Parathyroid hormone, thyroxine and estrogens are also needed.

PRELIMINARY EXAMINATION AND EMERGENCY CARE.

The mandible is fractured under many circumstances. A

child may fall while skating or riding a bicycle, adults will get into arguments or fall down stairs and today, one of the most common causes is the automobile accident.

Since there are numerous reasons for the injury, the problem is presented to the doctor in several ways. The patient may arrive, with a minimum of pain, with no lacerations or bleeding; complaining only of difficulty in opening and closing the mouth. Or they may be brought into the emergency room in severe distress. The severely injured patient must be examined promptly and given emergency care. The preliminary examination should survey the situation and determine what life saving immediate care is needed. The most urgent problems are respiratory obstruction or severe hemorrhage. Both may be present and perhaps complicated by unconsciousness.

Airway obstruction can occur for a variety of reasons and must be corrected immediately. When the patient is unconscious and supine, the tongue falls backward. If we add the additional displacement of soft tissues that occurs with the fractured mandible, the airway is closed.[7] Salivary secretions, vomited food or blood clots accumulated in the pharynx will cause further obstruction. A broken denture may be impacted there. An examination to determine the cause of the obstruction must be done. The tongue is grasped with a hemostat or sponge holder and pulled forward. The pharynx is inspected, and any foreign bodies which are present are removed. Secretions, vomitus and blood are aspirated. If the patient can be safely turned, and put in the prone position or on the side, this will help in maintaining an open airway. The jaw and tongue will fall forward and open the hypopharynx. When the patient cannot be moved, an oral airway or nasopharyngeal tube must be inserted.[7] If these measures do not relieve the obstruction and it continues, the larynx must be examined. Food or a piece of denture may be lodged between the vocal cords and must be removed. Occasionally, the patient, in addition to a fractured jaw, may also have a crushing injury to the larynx. The edema or a hematoma of the tissues may be causing an obstruction and must be recognized. The

airway may be established by inserting an endotracheal tube, a bronchoscope or by doing a tracheotomy.[8] If there is no evidence of laryngeal obstruction as the cause of respiratory distress, the chest must be examined. A pneumothorax, produced by a broken rib puncturing the lung must be recognized and relieved quickly. A 50 cubic centimeter syringe with a No. 16 needle can be used to withdraw sufficient air from the pleural space to allow expansion of the lung. For more lasting decompression, underwater drainage must be used, until the lung continues to remain expanded. The conscious patient with respiratory obstruction is apprehensive and uncooperative from hypercapnia.[9] Oxygen by face mask or a nasal catheter must be given to relieve the distress. No time can be lost in treating patients with respiratory difficulties since a few minutes of cerebral anoxia will produce irreversible brain damage.[10] When the patient's airway becomes progressively obstructed from edema, a tracheotomy should be done. The tracheotomy facilitates the removal of any aspirated material, and by passing a catheter into the trachea the patient can be given a high concentration of oxygen. The tracheotomy is valuable in handling the patient with a pulmonary injury or the aged patient with diminished pulmonary reserve.[11,12] Doing the procedure early enough may prevent future complications such as atelectasis and hypostatic pneumonia.[13]

Severe bleeding must be treated quickly or it will exsanguinate the patient. If conscious, they will try to remove the blood from the pharynx and mouth by coughing and expectorating. If unconscious, the blood will accumulate in the pharynx and enter the larynx. Profuse bleeding is frequently associated with the compound fracture, the tongue and the floor of the mouth. If the soft tissues are intact, the bleeding may stop as the hematoma enlarges, otherwise, the bleeding vessels must be clamped and ligated. The abraided areas require local pressure to stop the oozing. Occasionally, a hematoma may form at the base of the tongue or in the vallecula and produce respiratory obstruction. These are incised and drained when detected. The patient's blood loss is estimated by the hematocrit, and until it is replaced intra-

Fig. 1. The fixation halter is created by modifying the cervical traction halter. It can be made in three sizes for an accurate fit.

venous saline or plasma should be given.[14] When in shock the vasoconstrictor drugs neo-synephrine hydrochloride and Levophed Bitartrate are given by intravenous drip. In the aged patient with arteriosclerosis a sudden blood loss may be fatal, and therapy must be started quickly.

Pain is relieved by the injection of demerol or morphine; however, when there is respiratory obstruction or a brain injury, drugs which depress respiration such as morphine or barbiturates should not be used.

Cases with compound fractures should receive 3,000 units of tetanus antitoxin or if they have been previously immunized, one cubic centimeter of tetanus toxoid.

A survey should be made of the patient's associated injuries. The severe accidents frequently produce multiple injuries and they may not be readily detected. The abdomen as well as the skull, spine and extremities should be carefully screened.

Until a careful evaluation can be made of the fracture, the mandible should be immobilized. The Barton bandage may be used; however, the fixation halter can be applied (see Fig. 1). It is a modification of the halter used for cervical spine traction, and is made by substituting a buckle in place of the rings. Three sizes, small, medium and a large, can be prepared in advance and kept ready for use. With gauze padding in the chin for comfort, it maintains immobilization until time for surgery. In addition, the fixation halter is used in postoperative immobilization and can be substituted for the plaster chin bandage described by Schaefer.[15] The halter has the advantage of being easily removed for dressings and can be changed when soiled.

During the preliminary examination the injury to the mandible is put in its proper perspective, and the more serious injuries are given priority.

THE EVALUATION.

After the patient has received the necessary emergency care, the total problem must be evaluated. This requires detailed consideration of several factors.

The type and extent of injury to the mandible must be accurately established. Considerable information can be obtained by careful inspection and palpation. The examination starts with the region of the temporo-mandibular joint. Pain elicited on pressing over the joint may indicate a fracture of the head or neck of the condyle. Displacement of the condyle anteriorly indicates a dislocation, which may occur without a fracture. The ramus is palpated for crepitus, since dis-

placement in fractures of this structure are rare. The contour of the angle of the mandible can be palpated between thumb and forefinger and any changes detected. The body and symphysis are examined along the inferior border for any break in continuity. When a suspicious area is located, it can be further examined by holding the fragmented ends between thumb and forefinger of both hands and gently trying to produce movement. The patient is asked to open his mouth and any limitations or deviations of motion should be noted. A displaced fracture involving the neck of one condyle will produce a deviation of the jaw to that side on opening the mouth. The gingival and buccal mucosa are examined for lacerations. Fractures of the body are frequently compounded into the mouth and may be evidenced by a laceration of the gingival mucosa. The contour of the mandible is palpated within the mouth, on both the buccal and gingival surfaces, starting as high on the ramus as can be reached. Occasionally a severe facial swelling will disguise a fracture which can be detected manually on an intra-oral examination.

The dental arches are examined and loosened, broken or absent teeth should be noted and charted. The graphic method of charting used by dentists should be learned. The remaining teeth are palpated for rigidity to assess their value as anchoring pegs for wiring. The presence or absence of dentures is noted, and if absent the patient is asked whether they are available since the dentures, even if broken, are of value in determining the dental occlusion. The normal dental relationship must be determined, since this is the most valuable guide in the alignment of fragments, and all methods of reduction are based on the reconstruction of the patient's normal occlusion.

The mandible should not be examined as an isolated organ, but must be considered in its relationship to the maxilla. Both structures are involved in some of the methods of treatment and must be considered as one unit. If the mandible alone is fractured, the maxilla will serve as a splinting arch for wiring; therefore, in the thorough evaluation of the

fractured mandible one must also evaluate the condition of the maxilla. The same findings such as crepitus of bone, displacement of dental arches, injury to teeth and lacerations of mucosa are also noted for the maxilla.

The complete evaluation includes a radiographic study. Although the physician may feel confident of his findings on physical examination and believes that no additional information can be obtained radiologically, radiographs should be made. Some impacted fractures may not be evident clinically but will be detected on X-ray. Radiographs make possible a progressive study of the case and the early detection of complications. If litigation occurs a valuable record will be available.

The study requires several views. The postero-anterior (nose-forehead) position will visualize the condyle and the ramus. The occipital-mental (nose-chin) position will bring into view the coronoid processes and is also valuable for the maxilla and malar bone. The straight lateral view silhouettes the profile of the upper jaw, but because of the superimposition of the two sides of the mandible it may mask a fracture of the body. This fault can be corrected by tilting the head and plate 30 degrees from the horizontal and photographing each side. The condyle and the ramus will be exposed. This same 30 degree lateral position with the head rotated 15 to 30 degrees upon the spinal column, will give an excellent view of the body of the mandible from the angle to the canine tooth depending on the degree of rotation. Fractures of the alveolar ridge, particularly in the region of the incisors, may be clarified by using intra-oral films. Further views may be suggested by the radiologist for a specific case.[16] The radiographs should be considered an aid in diagnosis and should not be interpreted isolated from the patient.

After the local pathology has been evaluated, consideration is given to other factors. The type of anesthesia to be used is considered, and the less severe fractures can be handled with a local anesthetic. This may be given in the form of an inferior alveolar nerve block, unilaterally or bilaterally.[17] If

the fracture is in the midline, supplementary infiltration of the mucous membrane in the area may be necessary. If the wiring of the upper dental arch is required, the gingiva can be infiltrated with the anesthetic agent. Novocain 2 per cent with 1:100,000 epinephrine or Xylocaine 2 per cent with 1:100,000 epinephrine is used. The severe injuries will require a general anesthetic, and it must be determined whether the patient's medical condition will tolerate it. Many of our mandibular fractures are associated with multiple injuries, and some of these may be of more serious consequence to the patient. Surgery on the jaw should be deferred until the patient has recovered consciousness although a tracheotomy is valuable in removing accumulated pulmonary secretions. This concept was first developed by Galloway[18] in treatment of bulbar poliomyelitis and subsequently recommended by others.[19,20] If the patient is in shock from blood loss, replacement therapy is given and the blood pressure restored and stabilized. When more than one surgical procedure is required, interdepartmental planning will prevent the repetition of anesthesia. The orthopedist frequently can reduce a fractured femur, while the mandible is being repaired. Although the choice of the anesthetic agent is left to the anesthesiologist, the one most frequently used is intravenous pentothal and succinylcholine with endo-tracheal oxygen and nitrous oxide.[21]

The technique of administration will vary. When the patient has had a preliminary tracheotomy, the endo-tracheal tube can be passed through the opening. The surgeon will have to decide on the need of a tracheotomy postoperatively, particularly when wiring has been used and the jaws are closed. The use of the tracheotomy will prevent the postoperative asphyxiation from vomiting, which may occur before the wires or rubber bands are cut. At other times the patient can be spared a tracheotomy. The fixation of dental archbars can be done under general anesthesia, and later when the patient has reacted, the jaws can be fixed together with wires or elastics.[22] When the surgeon is in doubt as to the need of the tracheotomy, it should be done.[23] The risk of sudden asphyxiation is much greater than that of the surgery. The patient who has concomitant injuries of the chest and

who will have difficulty postoperatively in handling secretions will benefit from a tracheotomy.[11]

The patient's general medical condition will need to be evaluated with a careful history and physical examination. The elderly patient may have a cardiac, pulmonary or metabolic disorder which will require special attention from the internist. These conditions need to be controlled before surgery can be safely performed.

The evaluation of the patient extends beyond the pathology of his mandible, and plans must be made for the entire hospitalization.

METHODS OF TREATMENT.

A review of the literature of the past 50 years reveals how much thought and ingenuity has been devoted to this subject. It also becomes apparent that interest was stimulated by specific circumstances; the wars, the development of inert metals and the use of antibiotics. The methods of therapy changed as new developments occurred, and this process will continue.

Although many techniques of reduction and fixation have been devised, only two basic methods are involved: first, the use of the teeth for fixation either with direct wiring, archbars or dental splints; second, the direct approach to the bone with wire, plates, pins or external appliances. The experience of many surgeons has shown that no single technique is applicable in all situations because of the variety of injuries. After reviewing the methods, it appears that 80 to 90 per cent of the mandible fractures can be satisfactorily treated by either interdental or archbar wiring.[2,3,24,25,26,27,28,29] The remaining cases because of the absence of teeth or the location or displacement of the fracture must be handled by another method of fixation, and opinions differ as to the best approach. In skillful hands many of the techniques can produce equally satisfactory results.

In evaluating the techniques for the purpose of this paper, they had to fulfill special requirements.

1. The equipment should be simple to use.

2. A minimum of specialized laboratory facilities should be required.

3. The methods must be safe and reliable.

Each technique was evaluated and accepted or discarded.

Interdental Wiring.

This is one of the most useful methods of all. Although this technique was developed early,[24,30,31] with modifications,[32,33]

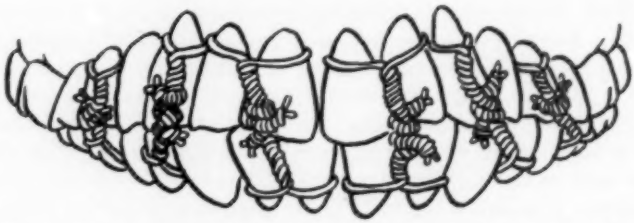

Fig. 2. The superior and inferior interdental wires are tightened with the teeth in normal occlusion, producing intermaxillary fixation.

it can be applied successfully in many cases. At first brass was used and later, stainless steel wire. Stainless steel wire is flexible, strong and can tolerate twisting without breaking. The finer grades such as No. 30 may serve, but No. 26, a heavier gauge, is more satisfactory. The wire is placed around the neck of the tooth and twisted firmly. The incisor teeth are best treated in pairs and the rest individually.[4] The attached wires on the opposing teeth are drawn together, bringing the dental arches into occlusion, and tightened. By firmly tightening several wires on each side of the dental arch, the jaws can be rigidly immobilized (see Fig. 2). Many variations of anchoring the wires to the teeth have been developed, and one of the most useful is the Ivy loop.[24] This method overcomes the objection of having to wire the jaws together at the time of placing the wires around the teeth. By using the Ivy loop technique, the teeth are first wired, and later the dental arches can be brought together with

wire or elastics. Kazanjian has recommended the use of the button technique, and it has been used successfully for many years.[4] The twisted portion of wire is rolled into a small button which is then used as a peg for holding elastic bands.

Interdental wiring is also used across the fracture line as a preliminary procedure to bring the fragments together, before the dental arches are wired together. When the mandible and the maxilla are wired together, the result is intermaxillary fixation.

Arch Bar Wiring.

This technique is second to interdental wiring in usefulness. The original arch bar of German silver has been varied to include loops (Jelenko splint) or buttons (Winter splint); or it may be made in a continuous row of malleable Monel metal (Erich splint) which can be cut to the necessary length. The Jelenko and Winter splints are semi-rigid, but they can be bent to fit the dental arch and shortened with a cutting pliers. The Erich splint is more flexible and can be molded to the shape of the dental arch by finger pressure. Both types of splints serve a special purpose. The rigid splint acts as an orthodontic appliance and gives greater stability to the fracture line. The flexible splint is useful in producing intermaxillary fixation with rubber bands or wire and is easier to apply.

The application of the splint consists of bending its shape to fit the dental arch and then wiring it to the available teeth. Number 26 gauge stainless steel wire is used for this purpose. Refinements in the fixation of the splint to the teeth have been developed.[2,3,4,28,29]

The arch bar is most valuable when one dental arch has only a few teeth present. It can be wired to the opposing teeth directly or to an opposing arch bar. Frequently both arch bars are brought together with elastic bands (see Fig. 3).

Dental Splints.

There are three main varieties of dental splints which are in common use. One of the earliest, and of orthodontic origin,

is the Angle splint. It consists of a gold band to which is soldered a gold bar. The band is placed around a tooth and fastened securely with a screw and a bolt. The bar is bent to the contour of the dental arch and then wired to the teeth.[4] The bands are made in various sizes to fit the different teeth. The Angle splint is useful when only one tooth is present on the posterior fragment; however, the same result can be accomplished by the proper application of a rigid arch bar.

The dental cap splints are used by those who apply the dental technique for all mandibular fractures.[2] Their use

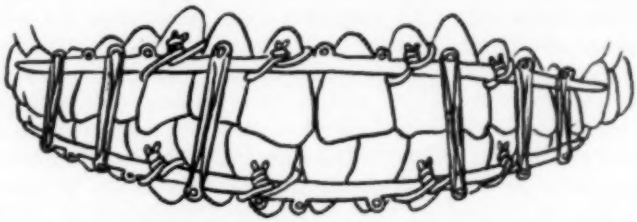

Fig. 3. The opposing arch bars are brought together with elastics or wire.

requires dental training and experience. As soon as possible after the injury, impressions are taken of the patient's teeth and from these the normal dental arch is reconstructed, usually in two sections. The splints are cast to fit the impressions and are then cemented to the teeth. The sections of the splint are fastened together with screws. An important prerequisite for the use of this technique is the availability of special laboratory facilities to construct the splint. When these facilities are not immediately available, time will be lost. Their application also requires the special knowledge of dental techniques, and this is not part of the otolaryngologist's training. Besides this, there are two other objections to these splints: the caps are cemented to the teeth, and this alters the patient's normal bite by the thickness of the cap. Over the period of weeks which would be needed for the fracture to heal, the teeth may be adversely affected by the pressure. In other instances, the cement may loosen, and

the splint will come off. It can be wired to the teeth for added fixation, but this is a duplication of effort. Most of the problems that would be handled by a cap splint can also be handled by the arch bar or interdental wiring.

The Gunning splint is used in association with alveolar or circumferential wiring.[2,3,4,28,29] It is made of an acrylic plastic and is lined with gutta percha. It can be prefabricated and kept ready in various sizes. When used, the splint is placed over the fractured dental arch, and by tightening wires placed circumferentially around the body of the mandible or through a hole drilled in the alveolar bone, immobilization can be given to the fragments. At least two wires are needed on either side of the fracture line. This splint without intermaxillary fixation does not always furnish complete rigidity, and a fracture behind the last molar tooth, cannot be immobilized with this method unless other measures are used, such as the head cap with traction.[34] The Gunning splint has its greatest usefulness in the small child, where injury to the unerupted teeth must be avoided.[35,36,37,38] It is held in place with circumferential wiring until healing occurs.

A modification of the circumferential wiring technique is used to immobilize a fractured edentulous maxilla to the mandible. A hole is drilled through the alveolar process of the maxilla, and the stainless steel wire which is passed through this hole is firmly anchored to the teeth of the mandible. The same process can be applied to the mandible if it is edentulous. The wires are removed when the fracture is healed.

Other variations in dental splints have been devised for specific purposes, but they do not alter the basic principle.[39,40]

Interdental or arch bar wiring can handle most of the cases treated with dental splints, and the remainder can be handled by other less involved techniques.

Internal Pin Fixation.

The use of the transfixion pin or wire of the Kirschner type to hold the fragments together has been described by

various authors.[41,42,43,44,45] Heavy Kirschner wires, bicycle spokes or round stainless steel pins have been recommended. In this technique, a wire is passed through the bony fragments with a drill. This produces fixation in one plane. The fragments, however, may still rotate or move laterally on the single wire. A second wire is usually applied and this eliminates the rotation; however, there may be some motion along the long axis of the pins, and by using interdental or arch bar wiring this can be prevented. This method has been used in a large variety of jaw fractures[43,45] including those of the condyle.[44] An objection to this method is that it must be combined with one of the other techniques for the most accurate alignment of the fragments; also, since one end of the wire is left protruding through the skin for the several weeks required to heal a fracture, there is a potential channel for infection to enter.

External Skeletal Fixation.

The method of external skeletal fixation introduced by Anderson[46,47] was applied to the fractured mandible by Converse and Waknitz[48] in 1942. This method received considerable evaluation during the last war[49,50,51,52,53,54] and again more recently.[55,56] Many variations of the basic appliance have been devised,[57,58,59,60,61] but the fundamental principle is the application of pins or screws into the bone which are then connected externally to a rigid bar. When the screws are fastened securely to the bone and to the bar, the fragments are immobilized; however, the screws have a tendency to work loose from the bone, and the immobilization will fail. The opening through the skin is an invitation to infection, and patients have complained that they cannot properly clean their faces and that they keep hitting the appliance. An important surgical objection is the difficulty in producing and retaining an extremely accurate alignment of fragments.

Open Reduction with Interosseous Wiring.

Since this method was first included in the treatment of jaw fracture its use has progressively increased.[27,62,63,64,65,66,67,68,69,70,71,72,73,74,75,76,77,78,79] A change has occurred from the time

Dunning[32] (1934) advised against the use of wire sutures in bone to the opinion of Gordon[65] (1947) and Plumpton and Crawford[73] (1955) who use this method in most of their problem cases.

This technique requires an exposure of the fragments, drilling small holes through them and fastening the pieces together with wire. The fragments can be aligned with great accuracy and by using several loops of wire, rigidity can be obtained. Earlier, No. 24 gauge brass wire was used,[63] then tantalum,[64,79] and now stainless steel wire[67,70,73] is preferred.

The fracture can be approached from either of two directions. Orally, the gingival membrane is incised, the periosteum elevated, and the ends of the fragments are exposed. Holes are drilled through the alveolar portion, wires are passed through and tightened and the periosteum and gingival membranes sutured.[28,64] The wires are permanently located beneath the gingival membrane, and they may prevent a comfortable fit with dentures.

The more familiar and preferred approach is through an upper neck incision. A 3.5 cm. incision is made a finger's width below the lower edge of the mandible over the fractured area. By separating the tissues horizontally with blunt dissection, injury to the mandibular branch of the facial nerve is avoided. Its location, described by McCormack,[80] is constant. The periosteum is exposed, incised and elevated from the fractured bone for a distance of one centimeter on either side of the fracture line. The fragment is held firmly with a clamp and while protecting the soft tissues under the fragments with a flat metal retractor, holes are drilled. It is important to remember the location of the inferior alveolar canal and to stay beneath this level. The No. 26 stainless steel wires are passed through the holes and tightened (see Fig. 4). The periosteum is closed with 3/0 chromic catgut suture and the skin with 3/0 black silk without drainage. Fractures in the condylar area can also be treated with open reduction and wiring either preauricularly[81,82,83,84] or endaurally.[85,86]

When there is a fracture compounded, externally or orally, this technique can still be used. If a comminuted fracture, those fragments with attached periosteum may still be viable and should be preserved. A thorough cleansing of the soft tissues with removal of all foreign bodies and debris is imperative. A soft rubber tissue drain is put into the area of the fracture, the periosteum is sutured and the skin closed. The drain is left in until all drainage stops.

The wires which are inserted are left in permanently. When the tissues have been thoroughly cleaned and antibiotics used,

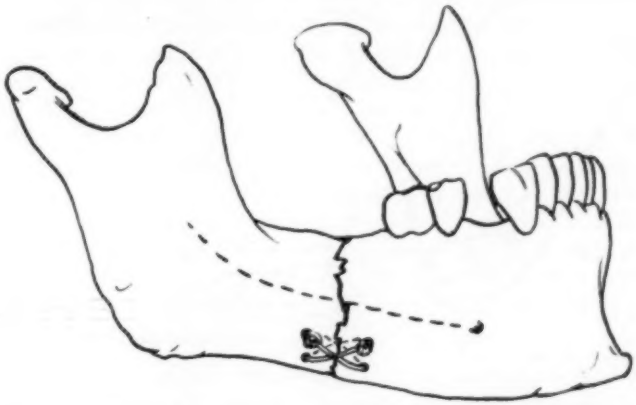

Fig. 4. Interosseous fixation is produced by drilling holes on either side of the fracture and then immobilizing the fragments with No. 26 gauge stainless steel wire. The mandibular canal must be avoided when drilling the holes.

removal of the wires because of infection is uncommon.[64,65,73] The frequent reason for removal has been the electrolytic action and this will be discussed later. Gibson[76] has devised a technique of removal of wire sutures which he recommends for compound fractures. The surgeon, using open reduction methods, should not become complacent because of antibiotics. The resurgence of resistant staphylococci emphasizes the continued need of obeying good surgical principles of debridement and cleaning.

Open Reduction with Plating.

This procedure is a development of the previous one. Instead of wire being used to approximate bone fragments, larger pieces of inert metal are fastened to the bone by wire or screws (see Fig. 5). The use of metal plates for fractures of long bones had been previously described[6] and vitallium plates were used to replace bone removed in surgery of the mandible.[87,88,89,90,91] Various authors[92,93,94,95,96,97] have described

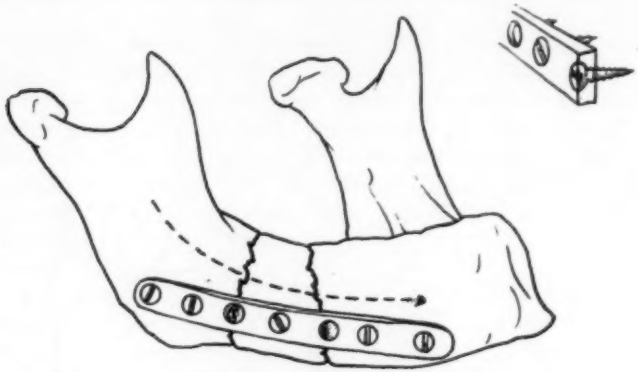

Fig. 5. The vitallium plate is fastened to the fragments with screws producing a rigid mandible.

the treatment of the jaw fractures with open reduction and plating, but it must be recognized that this technique is not used for the routine reduction of the mandible fractures but only in special circumstances. Severe fractures of the mandible with a loss of bony tissue, to anchor a bone graft, to make the mandible rigid in the combined fracture with the maxilla; these are indications for using the metal plate. All of the precautions mentioned under interosseous wiring apply to the use of the metal plates; however, they have been safely used in compound fractures immediately after the injury,[88] but temporary drainage is used in all of these cases. If there is no evidence of gross infection, the incision can be closed immediately after the insertion of the plate.

The use of open reduction with wiring or plating makes possible the treatment of the majority of mandibular fractures which cannot be satisfactorily reduced and immobilized by interdental or arch bar wiring.

The question arises as to whether there is any harm in the permanent retention of this metal.

TISSUE REACTION TO METALS.

With the acceptance of the open reduction technique, surgeons became interested in the cause of their failures. Reductions which were free of infection, were unsuccessful because the plates would not hold. The bone around the metal would break down, the screws would loosen, and the plate would slip out of position. Venable,[98] interested in this problem, determined that electrolysis was the principal cause of failure of metal appliances in bone. Dental research has also shown that metals in the presence of saliva act as a galvanic cell with erosion of the metal and that tissue changes result.[3] Metals vary in the electrolytic effect, and in a few this effect is minimal;[99,100,101,102] therefore, the maximum success in retaining metals in tissue occurs with the use of the most inert metals. These are divided into two groups: the alloys and the pure metals.

In 1926 the present 18-8S Mo type of stainless steel alloy was developed. In addition to iron, it contains chromium, nickel, silicon and molybdenum. This alloy can be machined, cast into plates and drawn into wire. It also has maximum corrosion resistance and a minimum of electrolytic action. Recently it has been further improved producing the 19-9S Mo alloy.[101] In small quantities, as is used in interosseous wiring, the effect on bone is minimal, but in the larger amounts of plating the electrolytic effect is increased.

A more inert metal was needed. This was developed by the Austenal Laboratories in 1929 and named "Vitallium." This alloy of cobalt, chromium, molybdenum, manganese, silicon and carbon is the most inert available and causes no appreciable electrolytic action.[99] It has no stimulating or

retarding effect on the healing of bone[102] and in several cases, infected wounds healed around the metal.[93] Venable, in a review of 1,227 cases, found that it can be left in the body indefinitely with no danger of late tissue damage.[99] Where it has been removed many years later, the metal and screws are bright and untarnished and the surrounding tissues are normal. Vitallium when introduced into the tissues immediately forms a chromium oxide surface film, and this prevents corrosion. Its remarkable inert qualities are slightly offset by its hardness, which requires that all plates and screws be cast; also, it cannot be drawn into wire and has only a small tolerance to bending.

Tantalum, a basic element, has a minimal electrolytic action. It is strong, tough, malleable, can be drawn into wire or machined, but it cannot be cast; however, it is available only in wire and skull plates.

The electrolytic effect is increased when two types of metals are used together. Chromium plated steel plates are unsuccessful, particularly when fastened with steel screws. Vitallium plates should be immobilized with vitallium screws and not stainless steel screws or wires. Although the silver alloy arch bar (Jelenko or Winter) is fastened to the teeth with stainless steel wire, a lesser electrolytic effect is obtained between a stainless steel arch bar and wire, and when applicable they are preferred.

The ideal metal for the purposes of the mandibular fracture would be as ductile and tough as stainless steel and as inert as vitallium or tantalum.[100] Research in metallurgy will someday achieve this goal.

REPAIR OF SOFT TISSUES.

The healing process begins soon after the injury, and repairs should not be delayed. Unless the soft tissues are approximated, granulations will fill in the defect and produce distortions and disfigurement.

Lacerations of the face overlying a fractured mandible are frequently ragged and dirty. There may be a large amount

of debris ground into the wound, and the severe compound fracture may resemble a war injury.[77] The longer the contamination is permitted to remain, the greater the danger of infection.[103] If the surgeon, after his evaluation, decides to proceed immediately, the cleansing of the injured area should be deferred until the patient is under general anesthesia. When the patient is unconscious and the major surgery is delayed, the laceration is irrigated promptly with warm saline and all obvious debris is removed. The tissues are approximated with a few loosely tied silk sutures, rubber drains are inserted and a dry dressing is applied. The reduction of the fracture is then deferred until the patient's head injury has been evaluated and treated. The conscious patient, whose reduction is postponed for some reason, should have the laceration repaired promptly. Irrigation is used and if debridement is necessary, the area is infiltrated with 2 per cent novocain with 1:100,000 epinephrine. It is preferable to do these procedures in the operating room, rather than in the emergency room.

The repair of the laceration, whether done with the reduction or alone, begins with cleansing.[104] The skin is washed with Phisohex or Septisol and foreign bodies which are imbedded are diligently picked out to prevent tatooing. The laceration is separated and irrigated with copious quantities of warm normal saline. Every hidden corner is inspected for a foreign body. The debridement should be conservative, although devitalized tissue must be removed. Ragged skin edges are made smooth and bleeding vessels are ligated.[105] Frequently the fracture is exposed and can be inspected. Devitalized bone fragments are removed, but if there is periosteum attached they are retained. If, in the judgment of the surgeon, interosseous wiring or plating will be required, it can be done at this time. The periosteum is important in healing, and effort should be made to preserve it. In the closure, the tissues are approximated with chromic catgut suture. One or more rubber tissue drains are inserted, and the skin closed with small interrupted dermalon sutures.

Unless noted on examination, oral lacerations may be over-

looked. Occasionally they are more extensive than those on the skin surface. When the buccal mucosa or periosteum is detached from the mandible, it is replaced as accurately as possible. The edges are approximated with chromic catgut, but space is left between the sutures to permit drainage.

Antibiotic or antiseptic solutions are no longer used to irrigate the wound because of their tendency to sensitize or destroy the tissue. Infection in the injured area is prevented by the use of systemic antibiotics.

FRACTURES OF THE MANDIBLE.

Condyle.

Condylar fractures are among the common fractures of the jaw. They are of two types: the intracapsular, with injury to the head of the condyle, are rare while the extracapsular or those that occur to the neck are frequently seen.[2,3] Among the neck fractures, we have those due to minimal trauma and no displacement; the greenstick type; and the fracture with displacement. The insertion of the external pterygoid muscle on the head, produces a displacement of the fragment when the fracture at the neck is complete. A dislocation of the condyle can occur without a local fracture, and when present there frequently is a fracture in another part of the mandible.[77] Thoma [82] and MacLennan[106] have thoroughly described all the varieties of fractures that may occur.

There are two opinions for the treatment of the condyle fracture. First: that all condylar fractures should have an anatomical reduction with immobilization.[81,82,83,84,85,86,107] Unless this is done, the occlusion will be altered, and eventually symptoms will arise in the injured area. The condyle is restored to its normal alignment by open reduction and wiring of the fractured ends,[81,82,83,84,85,86] the use of the Kirschner wire[44,45] or by plating.[23,97] The second opinion is treatment with immobilization alone for all types of fractures.[29,108,109] The mandible is immobilized by intermaxillary wiring or elastics, with the teeth in normal occlusion. A fibrous union occurs at the fracture site, which will function as a joint, even

if the fragments are displaced and not in alignment. Between these two views lies the contemporary opinion.[23,72,110] The majority of fractures can be handled adequately by intermaxillary wiring, a few will require an open reduction or a condylectomy.

A non-displaced, non-painful fracture of one or both condyles may need only a liquid diet and restriction of chewing for a month. A painful fracture either without displacement or a greenstick variety can be made comfortable with intermaxillary wiring or elastic band fixation. Sometimes the wearing of the fixation halter for one week will relieve the pain without interdental immobilization. The halter immobilization is useful in the edentulous patient with one fractured condyle. A unilateral displaced fracture of the neck, in a patient who has some teeth, is best treated by intermaxillary fixation. Either interdental wiring or arch bar wiring with elastic immobilization is used. The jaw is immobilized for four to six weeks in adults and three to four weeks in children. The intracapsular fracture with comminution of the head, is treated in the same way. When there are two fractures of the mandible with one at the neck, the condylar fracture is treated in the same manner; by immobilizing the mandible in normal dental occlusion after the other fracture has been reduced and immobilized. The majority of condylar fractures will be satisfactorily treated by this conservative technique; however, special circumstances may require an open reduction and this should be approached with caution.

In a bilateral condyle neck fracture with displacement, the mandible will be pulled posteriorly, and this will markedly distort the occlusion. If this cannot be permanently corrected by intermaxillary fixation, open reduction and interosseous wiring of one condyle as described by Shapiro[72] will prevent this deformity. It is used in conjunction with intermaxillary fixation. When the condyle fragment is so displaced that the mouth cannot be opened or closed, even after manipulating the fragments, open reduction is needed. When the fragment can be reduced, interosseous wiring is used, if the alignment does not hold then a condylectomy is done.[110]

The condyle can be approached from three directions. The preauricular incision described by Thoma,[81,82,84] Dingman,[83] and Richardson[23] directly exposes the condyle and the joint. Its main disadvantage is the proximity of the branches of the facial nerve which are consistent.[80] If the nerves are not severed by the surgery they may be injured with retraction. Henny[111] has described the submandibular approach, through an incision under the angle of the mandible. The masseter muscle is separated from the bone, and the condyle is exposed under the muscle. Visibility for interosseous fixation or condylectomy is good. The main branches of the facial nerve are avoided, since they lie superficially to the masseter muscle. The location of the mandibular branch of the facial nerve must be appreciated and avoided in the incision. Recently Rongetti[86] and Davidson[85] have described the anterior endaural approach to the condyle, and those otolaryngologists familiar with endaural surgery may prefer this technique. Open reduction and interosseous wiring of the fracture should be combined with some form of mandible immobilization preferably interdental or arch bar wiring. The use of plate fixation as described by Stuteville[97] is not needed, and the use of the Kirschner wire or external skeletal fixation has the disadvantages previously described.

Coronoid Process.

Fractures of the coronoid process are rare.[112,113,114] Since the coronoid lies protected beneath the zygomatic arch, these fractures are seen combined with fractures of the arch and the malar bone. The fractured coronoid has minor clinical significance since the function of the detached temporal muscle is compensated for by the masseter and pterygoids. The most common symptom is pain rather than interference of function.

It is treated with intermaxillary fixation by applying arch bars and rubber bands or in the edentulous patient a head bandage of the halter type. When an open reduction is needed, and this would be a rare occasion, the submandibular approach described by Henny[111] would give a safe exposure to the area.

Ramus.

Although these fractures occur more frequently than those of the coronoid they are still uncommon.[112,113,114] Laterally a large area of the ramus is protected with the masseter muscle; medially, the internal pterygoid. The weakest point in the transmission of the blow to the jaw is at the neck of the condyle, and this fractures first. When fractures of the ramus occur, they are not displaced because of the overlying muscles. The treatment consists of using either dental fixation or a head bandage for immobilization. Open reduction is rarely necessary; however, the approach described by Henny[111] is excellent.

Angle.

Where the ramus joins the body of the mandible, there is a line of structural weakness, and here fractures occur frequently.[3] The presence or absence of posterior molar teeth, the direction of the line of fracture, the displacement from muscle pull have been responsible for a variety of methods of reduction and immobilization; however, the problem centers around one important point and that is the presence of displacement of the fragments.

The fracture which is horizontally and vertically favorable with no displacement of fractured ends, is satisfactorily treated by immobilizing the jaw with intermaxillary fixation in occlusion. If there is an associated fractured condyle on the opposite side, this will also be treated at the same time. When the fracture is unfavorable, either horizontally or vertically, intermaxillary fixation alone is not adequate since the posterior fragment is displaced.

It has been in treating this displaced fragment that many techniques have been devised for maintaining the reduction.[2,3,4,28,29,43,48] Among them is the use of open reduction and interosseous wiring.[68,72,73,74] This approach has simplified the problem and makes possible the treatment of all types of displacement with one method. The technique is simple and safe. A 3 cm. incision is made a finger's width below the angle of the jaw adjacent to the fracture area. The skin and

platysma are separated and the periosteum exposed. This is incised and elevated from the bone on either side of the fracture line. Two small holes are drilled through the bone on both sides of the fracture, and 26 gauge stainless steel wire is then passed through the holes and tightened, bringing the fragments together with great accuracy (see Fig. 4). The incision is closed without drainage, and the teeth are fixed together in occlusion to complete the immobilization. In a bilateral angle fracture this procedure is done on both sides, and the wire is left in permanently.

This method is especially applicable in the edentulous mandible. If adequate immobilization cannot be obtained with the use of the wire alone, the head halter can be used to supplement the fixation.

Body.

Fractures of the body are of two types: those with teeth on the posterior fragment, and those without. The latter type can be considered as being the same as the angle fracture and is easily treated with open reduction and interosseous wiring. The anterior fragment is then immobilized with intermaxillary fixation. If there are teeth in the posterior fragment, interdental wiring across the fracture line combined with wiring to an arch bar, followed by immobilization by intermaxillary fixation is usually satisfactory. A double fracture on one side, if there are teeth in the central fragment is treated in the same way.

In children, a non-painful and non-displaced fracture can sometimes be treated by using the fixation halter.

The edentulous mandible, with a single fracture can frequently be satisfactorily treated with interosseous wiring and immobilization with the fixation halter. The patient should be kept on liquids for three to four weeks. This type of reduction is extremely useful in the aged since early restoration of function occurs. When there has been marked comminution of fragments, and these cannot be firmly realigned with wiring, plating with a vitalium plate across the fracture line gives firm support to the separation. When combined with

Fig. 6. A displaced fracture of the right angle is combined with a comminuted fracture of the left body.

the bandage early restoration of function occurs. Bilateral fractures are also treated with wiring or plating as necessary and supplemented with intermaxillary wiring or halter (see Figs. 6 and 7).

Symphysis.

These fractures may be a true separation in the midline or they may be oblique. When teeth are present on either side of the fracture, interdental wiring across the fracture line and then fixation to the maxilla will give good immobilization. If only a few teeth are present, then wiring to an arch bar with intermaxillary fixation will serve.

In the edentulous patient the use of open reduction with

Fig. 7. The angle fracture is reduced and immobilized with interosseous wiring and is combined with plating of the body fracture. The mandible is then immobilized to the maxilla with intermaxillary wiring.

interosseous wiring, through a submental incision is satisfactory. With a severely comminuted fracture, plating may be needed.

Alveolar Process.

When there is a fracture through the alveolar process with displacement of teeth, an attempt should be made to save the teeth if possible. The alveolar bone with the teeth attached, should be reduced to the normal occlusal position and retained by wiring to an arch bar. If the teeth have been avulsed by the blow, the alveolar bone should be inspected for roots and these should be removed by the dentist. When the alveolar bone is rough due to the fracture an alveolectomy is

Fig. 8. A fracture of the mandible is combined with a displaced fracture of the maxilla. (LeFort I.)

done. This procedure is performed by the consulting dentist at the time of surgery and with experience can be done by the otolaryngologist.

Fracture of the Mandible and Maxilla.

This type of injury is a more complicated problem than has been previously discussed. When the mandible alone is fractured, the maxilla can serve as a splint, to which the mandible is fixed[28] When both the maxilla and mandible are loose neither can serve as a splint unless one is made rigid. The

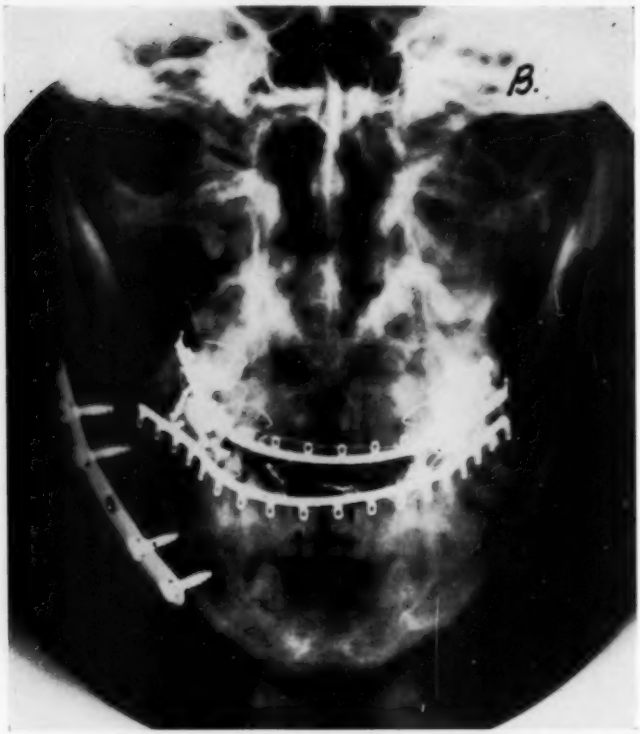

Fig. 9. The mandible is plated for rigidity and by applying arch bars with intermaxillary wire fixation, the maxilla is immobilized.

maxilla can be anchored by open reduction and wiring to the rim of the orbit or to the zygomatic arch as described by Adams and Walden.[66] As an alternative method, the mandible is created into a rigid splint, and the maxilla is then fixed to the mandible.[71] As long as good dental occlusion is maintained, the functional result will be satisfactory.

When the maxillary injury consists of a depressed fracture of the anterior wall without motion or displacement of the dental arch, intermaxillary fixation with arch bars will be adequate to fix the mandible. If the patient has an edentulous

Fig. 10. A bone graft taken from the iliac crest is used to fill in the defect and is held in position by the vitallium plate offering immediate rigidity to the mandible.

maxilla the mandible is immobilized by using the pyriform crest as described by Thoma[115] and Fordyce[116] or the nasal spine.[117] When the maxilla is displaced or freely movable it is then necessary to convert the mandible into a splint. This is done with the use of open reduction and either interosseous wiring or plating (see Figs. 8 and 9). If the mandible can be made rigid with the use of interosseous wiring rather than plating, this is preferred. Although the plates may be left in permanently without harm, it is preferable to use as little metal as is necessary to produce rigidity.

Fractures with a Loss of Bone Tissue.

Severe fractures of the mandible, particularly those which

are compound, may involve a loss of bone tissue. After the soft tissues have been cleaned, the bone fragments are inspected. Those which have no attached periosteum will probably die and act as a sequestrum unless removed. All those which are attached to the periosteum should be saved, since they may unite. If a loss of bone tissue has occurred and there is a space between the fragment ends, the final union will be fibrous and unstable and in these circumstances the defect must be filled in with bone for a satisfactory union. Several methods have been described using bone chips to fill in the defect.[4,28] The mandible is first immobilized to the maxilla with intermaxillary fixation and then through a submandibular incision the fracture site is exposed and the defect filled in with bone chips.

The vitallium plate is useful in this type of problem. Fig. 10 illustrates the use of the vitallium plate to hold in position a bone graft from the iliac crest until bony union has been achieved. The plate is still in place and has not caused any difficulties requiring removal. The plating was supplemented with intermaxillary fixation.

The bone graft is not inserted while there is evidence of infection in the wound. The laceration is allowed to heal while the fragments are retained in the occlusal position by interdental wiring. When there is no evidence of suppuration, induration or pain, the area is reopened with a submandibular incision, and the bone graft is inserted. The patient is maintained on antibiotics during the period of healing, before and after bone graft is done.

SUMMARY OF TREATMENT.

Fracture	Treatment
Condyle—	
1. A non-displaced and non-painful fracture.	Liquid diet and restriction of chewing.
2. A painful fracture with no displacement or a greenstick variety.	Intermaxillary wiring or rubber band fixation or halter immobilization in the edentulous patient.
3. Unilateral displaced fractures in patient with teeth.	Intermaxillary fixation with wires or elastics for 3-4 weeks (child) or 4-6 weeks (adult).

Fracture	Treatment
4. Bilateral displaced fractures in patient with teeth.	Intermaxillary fixation with wires or elastics. If unsatisfactory occlusion results then an open reduction and interosseous wiring of one side is used in combination with intermaxillary fixation.
5. Unilateral displaced fracture—edentulous patient.	Replace dentures in mouth and immobilize with halter fixation.
6. Bilateral displaced fracture—edentulous patient.	Halter fixation. If the jaw motion is restricted or marked displacement is present then use a unilateral open reduction and interosseous wiring combined with the halter.
7. Condyle combined with a fracture in another site in a patient with teeth.	Reduce and immobilize other fracture by interdental wiring or open reduction and then use intermaxillary wiring to immobilize condyle.
8. Condyle combined with a fracture in another site—edentulous patient.	Other fracture is immobilized with open reduction and interosseous wiring and the jaw is immobilized with the halter.
Coronoid Process—	
1. Patient with teeth.	Intermaxillary fixation by arch bars and elastics.
2. Edentulous patient.	Head halter for immobilization.
Ramus—	
1. Patient with teeth.	Intermaxillary fixation by arch bars and elastics.
2. Edentulous patient.	Head halter for immobilization.
Angle—	
1. No displacement of fragments—patient with teeth.	Intermaxillary fixation by wires or elastics.
2. Displacement of fragments (horizontally or vertically unfavorable) in patient with teeth.	Open reduction and interosseous wiring combined with intermaxillary fixation by wires or elastics.
3. Displaced fragments in the edentulous patient.	Open reduction and interosseous wiring or plating (if comminuted) combined with halter immobilization.
4. Bilateral angle fractures.	Open reduction and interosseous wiring combined with intermaxillary fixation by wires, elastics or head halter if edentulous.
Body—	
1. With teeth on the posterior fragment.	Arch bar immobilization and intermaxillary fixation with wires or elastics.
2. No teeth on the posterior fragment.	Open reduction and interosseous wiring combined with intermaxillary fixation with wires or elastics.
3. Edentulous mandible.	Open reduction with interosseous wiring or plating combined with halter immobilization.

Fracture	Treatment
4. Severe comminution of fragments with or without teeth.	Open reduction with interosseous wiring or plating combined with intermaxillary fixation by wiring or elastics or if edentulous, halter immobilization.
5. Double unilateral fracture with teeth in fragments.	Arch bar wiring and intermaxillary immobilization.
6. Double unilateral fracture — edentulous fragments.	Open reduction with interosseous wiring or plating. Immobilization with the head halter.
Symphysis—	
1. Teeth present on both sides of the fracture line.	Interdental wiring or arch bar wiring across the line of fracture combined with intermaxillary fixation with wires or elastics.
2. Teeth present on only one side of fracture or if edentulous.	Open reduction and interosseous wiring combined with intermaxillary elastic or halter fixation.
Alveolar Process—	
1. Teeth present in fragment.	Immobilize the teeth to an arch bar.
2. Edentulous patient.	If periosteum is attached retain the fragment, if not, then do an alveolectomy.
Mandible and Maxilla—	
1. Teeth present in mandible and maxilla.	Mandible is made rigid with arch bar wiring and then maxilla is immobilized to the mandible with intermaxillary wiring.
2. Teeth present in maxilla-edentulous mandible.	Mandible is made rigid with open reduction and wiring or plating and maxilla is fixed to mandible by use of alveolar process circumferential wiring of the mandible.
3. Teeth present in mandible with an edentulous maxilla.	Mandible is made rigid with arch bar wiring and maxilla is fixed to mandible with alveolar process circumferential wiring.
4. Edentulous mandible and maxilla.	Mandible is made rigid with open reduction and plating or wiring and the maxilla is fixed to the mandible with alveolar process circumferential wiring with the dentures between the arches to maintain the proper spacial relationship.
Fracture with a Loss of Bone Tissue—	
1. Fragments contain teeth.	The dental arch relationship is retained by intermaxillary wiring of the fragments until a bone graft is inserted.
2. Fragments are edentulous.	The arch relationship is maintained by bridging the gap with a metal plate until the bone graft is retained in proper position by interosseous wiring or plating.

FRACTURES IN CHILDREN.

The incidence of the fractured mandible in children is low. It is estimated that they occur in two to five per cent of all mandible fractures[35] and less than one per cent are in children under six years of age.[36] Since by the twelfth year the mandible closely resembles that of the adult except for the third molar tooth,[35] we consider this age to be the upper limit of the childhood fracture.

The child with the broken jaw presents a somewhat different problem from that of the adult, starting with the examination. The fear and lack of cooperation usually prevents a thorough physical examination and X-rays assume a greater importance. Although many fractures in the adult can be treated with local anesthesia it is almost always necessary to give the child a general anesthetic.[37]

The mandible of the child presents special characteristics. The bone is more elastic and softer, and this appears to predispose to a greenstick type of fracture, frequently of the neck of the condyle.[36] The presence of tooth follicles in the bone produces a potential weakness and may account for the widespread fractures that occur. The fracture lines in children may involve two to 14 teeth and a larger proportion of the bone than in the adult.[35] The presence of a growth center in the condyle, which may be injured, can lead to a deformity in the development of the jaw,[36,118] and this has been shown experimentally by Sarnatt.[119] Interference with the tooth follicles will prevent the normal development of the dentition.

The treatment of the fracture is based on the principal of using a method which will give minimum interference with the tooth follicles. Although some[118,120] believe that adult methods are applicable in most cases, many authors prefer special methods.[15,31,35,36,38,121,122] When the permanent teeth are present, intermaxillary fixation with arch bar and elastics is preferred. The arch bar is wired to the available secondary teeth as in the adult (see Fig. 3). The deciduous teeth, because of their shape do not lend themselves to the dental wiring methods or to the use of cemented cap splints,

and because of the presence of the secondary tooth follicles, some authors do not recommend the use of external skeletal fixation or open reduction with interosseous wiring.[15,36,38,121] Others believe that open reduction can be done when needed, but the unerupted teeth should be retained and the interosseous wiring be placed in the cortical bone without interfering with the developing teeth.[35,120] A fracture of the body behind the last molar tooth, the angle and the ramus can be safely wired by open reduction when necessary, as in the adult. Most surgeons recommend that condyle, neck and head fractures in children be treated conservatively by intermaxillary dental fixation or by a head bandage. The fixation halter described earlier serves adequately. A displaced fracture will need the specialized dental technique in the construction of a Gunning type of splint or an acrylic splint. The fractured segments are aligned under the splint, and the splint is then fixed to the mandible by circumferential wiring as described by Thoma[28] or Erich.[29,34] The mandible should be immobilized by fixation to the superior dental arch or by immobilizing the jaw with the fixation halter for additional rigidity.

FRACTURES IN THE AGED.

Medical progress in extending the life span has increased the number of aged patients. Kulowski[123] in his series of 661 automobile accidents reported that 11 per cent of his casualties were 60 years or older. We can expect that this group will continue to increase.

The patient who is 65 years old presents several interesting characteristics. The mandible may be edentulous and as a result of the absorption of the alveolar bone, rather narrow. When this is combined with the increased brittleness of the bone and the exposed position, it is surprising that it is not fractured more often. The older patient frequently has another illness for which he is or should be receiving therapy. A coronary sclerosis, hypertension or a pulmonary emphysema can significantly alter the prognosis of a fractured jaw.

The evaluation of the fracture must consider two aspects: the local problem, and the patient's general condition. Occa-

sionally it may be necessary to compromise a perfect surgical result for safety to the patient. The surgery should be deferred until the patient has had a general medical evaluation. and any necessary treatment. The choice of an anesthetic must be carefully considered and when possible local is used. The presence of excessive secretions in the airway of the aged patient may require a tracheotomy.[12]

Kennedy[124] in discussing the management of injuries in the aged has advised that function be restored as soon as possible. Older patients have a small tolerance for cumbersome external appliances and head caps. Because of these various factors the treatment of mandible fractures in the aged is made as simple as possible. When teeth are present intermaxillary wiring is used and if this cannot be easily or safely done, open reduction with interosseous wiring or plating is done. This latter method gives a rapid restoration of function. The postoperative course frequently requires special attention to the patient's nutritional and mental state. A high vitamin, protein and caloric liquid diet is prescribed, and the agitated patient with cerebral arteriosclerosis is made comfortable with the use of tranquilizers. Early mobilization will prevent complications and the patient should be permitted to sit in a chair as soon as possible.

POSTOPERATIVE CARE.

The nutrition of the patient is an important adjunct in the therapy. A high protein, calorie and vitamin liquid diet is prescribed and it can be taken by a straw or from a spoon. Where complete immobilization is not required, as in certain impacted fractures, soft solid foods such as cooked cereals, creamed soups and ice cream can be added. In children and in the aged the dietary requirements need additional attention. The child's weight should be checked periodically to be sure that growth needs are being met. The old patients may require dietary regulation for diabetes, cholecystitis or other medical conditions.

The use of antibiotics has made the open reduction technique safe and uncomplicated. Fractures in the areas with

teeth are usually compounded into the mouth. The same occurs with lacerations of the face which lie over the fracture. Both types of injuries can heal with primary intention when the antibiotics are used. When face lacerations are present the area is drained with a soft rubber drain for 48 to 72 hours to remove the accumulated blood and serum. Procaine penicillin 300,000 units and .5 grams of streptomycin are given twice a day for one week or until the soft tissue lacerations are healed. If there is suppuration, the drain is left in and the patient kept on antibiotics until it stops.

Mouth care is important in the treatment. Warm saline rinses after and between meals help to keep the area clean, especially when lacerations are present. The use of hydrogen peroxide or perborate solutions may produce undue tissue damage. The antibiotic solutions may sensitize the patient and are not recommended. The use of elastics for fixation enables a periodic examination of the mouth and cleansing of the teeth when indicated.

The postoperative care does not end with the patient's discharge from the hospital. They should be seen for intermittent follow-ups for several months which makes possible an early recognition of any delayed complications. If the patient has suffered dental injury he should be suitably referred. The surgeon's judgment will develop from observation and comparison of his results over a period of years.

SPECIAL EQUIPMENT.

The special equipment needed to apply the techniques outlined is easily available.

1. Stainless steel wire (alloy 18-8S Mo) in No. 26 and No. 28. This is available through surgical suppliers.

2. Wire cutter and pointed pliers. The types obtained from electrical supply houses are satisfactory.

3. Jelenko or Winters arch bars are available as a package unit from dental supply houses or directly. Jelenko Arch Bar: J. F. Jelenko & Co., Inc., New York 19, New York; Winters Arch Bar: Baker & Co., Inc., 113 Astor Street, Newark, New Jersey.

Small elastic bands for immobilization are included or they can be easily made from rubber tubing.

4. Erich Monel arch bar is available in a roll from: N. I. R. O., 280 West State Street, Westport, Conn.

5. Vitallium plates and screws are obtained from: Austenal, Inc., 224 East 39th Street, New York 16, New York.

Vitallium Bone Plate No. 6913 Mandibular 3" straight.

Screws, No. 6912—7/64".

Venable Screwdriver—7/64".

Vitallium Finger Plates, No. 6315—¾" and 1" with four holes.

Screws, No. 6349.

Screwdriver, No. 6884-3.

6. The drill for the interosseous wiring and plating can be either a Zimmer or Stryker drill and is usually available in hospitals. A drill bit size No. 55 or .052" is used.

SUMMARY.

The subject of the fractured mandible has been discussed as a problem to the otolaryngologist, the pertinent literature has been reviewed and various techniques described, evaluated and applied.

A method of treatment has been presented which can be satisfactorily applied by the modern otolaryngolgist trained in surgery of the head and neck.

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CORRECTION.

In the paper "Carcinoma of the Tonsil" by Dr. John F. Daly and Dr. Milton Friedman published in THE LARYNGOSCOPE, May, 1960, there is an error on page 606 in the last sentence of the first paragraph. It should read "The accuracy of the rotation is checked on an X-ray film in a phantom of the head and neck at the level of the lesion."

The last paragraph on page 606 should read "Cancer of the tonsil is one of the more radio-sensitive cancers of the upper respiratory tract and consequently lends itself to treatment with external radiation alone."

THE INFLUENCE OF DEAFNESS ON THE CREATIVE INSTINCT.*

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It has been said of the creative artist that his work, be it prose or poetry, painting or sculpture, music or drama, is the product of an inner compulsion whose only standard is of his own making; and that the desire to please others in no way influences or even stimulates his compositions, which, no doubt, accounts for the fact that many creative artists never live to see their work appreciated.

Bach in his own lifetime was scarcely known outside his own country, while Van Gogh sold only one picture, and that for but a few francs. A genius is not concerned with his impact upon society. He can only obey the creative impulse which sooner or later leads to immortality. It is interesting. therefore, to speculate upon the extent to which the outpourings of a man of genius can be influenced by his mental and his physical state.

The Pathetique Symphony of Tchaikowsky, first performed but three weeks before his death, expresses, particularly in its final movement, the tortured and perhaps over-dramatized despair of the neurotic, while the macabre and often malevolent heroes of Dostoevsky are reflections of his own character and circumstances.

My purpose here is to show how deafness may be said to have influenced the creative instinct of artists in three quite different fields: a writer, a painter, and a composer. In each I believe the cause of the deafness was different; in two it must surely have left its effect on their work, but it may well be disputed whether it had any effect on the output of

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the third; but as I shall hope to show you there may well be a good reason for this.

Man, as we know, is a social animal. He likes to talk, and he is occasionally prepared to listen; so that if he is deprived of his hearing he feels left out, and indeed he gradually gets left out of things. He becomes withdrawn and even suspicious, and one of my patients once told me that "deafness takes all the colour out of things"; which leads me to my first patient—the Spanish painter, Goya, who became suddenly and totally deaf in 1793 at the age of 47 as the result of a sudden and severe illness, which for a time affected his vision as well. This tragedy overtook him not long after he had been appointed Court Painter to Charles IV of Spain, at a time when he was in great demand as a portrait painter, though his reputation as a painter was founded on his compositions of Spanish life, all in the most delicate, though at the same time striking, tints of lemon, red, yellow and pale green. These colourful scenes were for the most part full of action, life and gaiety, and in this period of his activity one cannot but be impressed by his genius for depicting the pleasant side of Spanish town and country life.

Soon after he was overcome by deafness, Goya's artistic output changed, both in colour and in character. Much of the colour left his pictures; in fact the walls of his house, the "Quinta del Sordo" were covered with what, because of their sombre tints, are known as the "Black Paintings." Most of these have been preserved and can now be seen in the Prado Museum in Madrid, where they provide such a striking contrast to his colourful and gay earlier paintings, many of which are also to be seen in the Prado. Not only have they lost their colour, but they have also lost their gaiety. Instead, they dwell on the macabre and even at times the disgusting side of life. His series of etchings on the Horrors of War and the Capricios are curiously unpleasant. Though they may be true of the times and certainly of wartime, they are entirely different from what he produced in his earlier and healthier days. His social and political conscience seems to have developed to the extent of influencing his artistic output only after he became deaf.

At one time I wondered whether the unusual disorder that ushered in his deafness could have affected his appreciation of colour, but this surely was not the case, because he could on occasion conjure up the old colours, as can be seen in his last painting, "The Woman of Bordeaux," painted 25 years after his illness when he was approaching 80 years of age; or the famous though heartrending "Night of May 3rd"; or again the amazing and revealing Group of Charles IV and his family.

What then was this illness that for a time affected his vision and left him totally deaf? Of course, syphilis has been blamed, and there can be no doubt that, as the statistician would put it, he was often "at risk." Though happily and fruitfully married he had many love affairs and one of his models, who also was his mistress, by her beauty attracted the attention of the Prime Minister Godoy, who took her over from Goya in return for forgiving a political exile. Godoy himself was the lover of the Queen whose two youngest, and certainly as portrayed by Goya, brightest children, are said to have been fathered by Godoy.

Goya was also for a time lover of the Duchess of Alba, who, it has been said, was the model for his two paintings, the clothed and the unclothed "Maja."

No matter how hard he tried to acquire or avoid a specific infection, it is unlikely that this would suddenly attack vision and hearing and also, no doubt, balance at the age of 47, allowing full vision to return after a few months.

To me it sounds much more like a curious and little known affection of the pigmented areas in the head and neck in which temporary patches of white hair and white skin are accompanied by temporary affection of the uveal tract, and alas permanent loss of hearing and often of vestibular function. Known as the Vogt-Koyanagi syndrome, I have seen three full-blown cases and two other partial ones. In my cases the deafness was almost complete, and so far as I know, permanently leaving the patients in a sorry plight. I can well imagine what a profound and shocking effect this might have on a sensitive and gregarious artist. To be cut off from

his fellow creatures would encourage his creative instinct to turn towards the sorrows and even the horrors of life instead of its pleasures, and in those times of a corrupt court, invasion and domination by Napoleon Bonaparte, there were plenty of horrors to depict.

Let us now go back almost a century to study the case of another creative artist, this time the writer Jonathan Swift, who died at the age of 78 in the year 1745, just a year before Goya was born. Poet, wit, satirist, political pamphleteer, friend of kings and queens, Dean of St. Patrick's Cathedral, Dublin, but never, to his mortification, to become a bishop. He also was famous as a platonic lover whose correspondence with Vanessa and with Stella has entertained posterity more than it satisfied his languishing correspondents, who both died spinsters within a few years of one another and more than 25 years before the object of their unrequited or at any rate unsatisfied affection.

His introduction to political and court life came early as secretary to Sir William Temple, who played an important part in cementing the friendship between Holland and England and who had helped to bring William of Orange as joint occupant of the English throne with Mary.

Swift leaned towards the Tory side of politics which was under the guidance of Edward Harley, after whom Harley Street was named, and who later became the Earl of Oxford. The Tories were opposed to Marlborough and his wars, and Swift was their most potent and most strident pamphleteer.

His two most famous works, "The Tale of a Tub" and "Gulliver's Travels" were brilliant but biting satires on the times in which he lived, and later in his prose and his verse, he became at times venomous and even disgusting. One cannot help comparing some of his later works with some of Goya's drawings, both of them dwelling, almost harping, on the worst side of human nature and the times.

Towards the end of his sad life, Swift's days were darkened by deep depression which was regarded as madness, though

there is no real evidence to show that he had lost more than two of his senses, those of hearing and balance.

For this unfortunate man suffered from bilateral Meniere's Disease and was subject from early manhood to bouts of giddiness and deafness which pursued him throughout his life. Let us hear what the patient himself has to say about it all:

"I got my giddiness in 1690 (at the age of 23) by eating 100 golden pippins at a time at Richmond. Four years later at a place 20 miles further on in Surrey I got my deafness; and these two friends have visited me one or other every year since; and being old acquaintances have often sought fit to come together."

It is, however, probable that he was hard-of-hearing in one ear before the giddiness started because he wrote to his friend, Sheridan, as follows:

"In England, before I was 20, I got a cold which gave me a deafness that I could never clear myself of. Although it came but seldom and lasted but a few days, yet my left ear has never been well since; but when the deafness comes on, I can hear with neither ear, except it be a woman with a treble or a man with a counter tenor."

Clearly he was describing bilateral Meniere's Disease with severe deafness in the left ear and fluctuating low tone deafness in the right ear.

A sympathetic friend, Mr. Forde, gives us some idea of the view held then by patients of doctors who were trying to cope with a disorder which defied their efforts:

"These sort of disorders provoke the Physicians everywhere; and they are merciless dogs in purging and vomiting to no purpose when they know not what to do."

Poor Swift continues to protest against the purgings and bleedings ordered by his doctor, maintaining that his trouble lay neither in his belly nor in his blood, but in his head; and in this, of course, he was quite right.

After he died in 1745, the skull and contents were examined,

and it was stated by a Dr. Delany that the brain was loaded with water. This, in view of our present knowledge of Meniere's Disease, was not very far from the truth.

Here then we have a great writer, plagued all his life with giddiness and deafness from recurring bouts of Meniere's Disease: a depressing prospect for anyone; but for a sensitive creative and ambitious man, who better than any of his contemporaries could see and portray the follies of his time, and who was frustrated in his ambition to become at least a bishop, it is no wonder that his genius was soured. Whether his attitude to women was influenced by his disorder is a matter of conjecture. There can be no doubt that he enjoyed their company and that they were his for the asking; but he never asked. I have known many sufferers from this and from other forms of deafness who never married, and though I have never asked them outright, I have the impression that they did not want to saddle a partner with having to share such an affliction.

Here then are two great creative artists, each gregarious and each shut off from his fellow creatures by a severe form of perceptive deafness. In each the artistic output was tinged with pessimism and even despair; and I suggest that it was their deafness that played a large part in their gloomier outpourings.

Now we come to our third patient, the composer Beethoven, who was born in 1770 and who, despite his deafness must surely be the greatest composer the world has known.

Our first knowledge of his affliction comes from the Heiligenstadt Testament which was in fact his Will and was only opened and read after his death.

During the summer of 1802, Beethoven was in the small village of Heiligenstadt not very far from Vienna, engaged in composing his Second Symphony. He was 32 years of age at the time and his Will and testament which he wrote during this summer included the following remarks about his deafness:

"But only think that during the last six years I have been in

a wretched condition; rendered more by unintelligent Physicians, decreased from year to year with hopes of improvement, and then finally forced to the prospect of lasting infirmity."

Here then is the patient's own account of his deafness which he had noticed for at least six years—at the age of 26—and no doubt it had been coming on slowly before that.

Is there any wonder that Beethoven was a difficult man; one who could claim to be the very Prince of Sound and yet he could not hear himself?

All the deaf are sensitive about their affliction, but how much more sensitive a musician must be to the sly laugh or grin that his defect would be bound to evoke from the less charitable of his acquaintances; yet despite this great and progressive handicap he composed 100 more works after writing this Heiligenstadt Testament.

Towards the end of his life he was extremely deaf. He conducted the first performance of his Ninth "The Choral" Symphony, and as was the custom in those days, he was seated in a revolving chair. As the symphony finished he waited, still facing the orchestra, for the applause. There was dead silence. He looked at the orchestra, shaking his head, the tears pouring down his cheeks. Then they turned him around to face the audience who were cheering and clapping in a frenzy of applause which he could not hear.

After his death in 1827 his skull and temporal bones were examined but nothing definite was found, though as usual that etiological whipping boy of those days, syphilis, was evoked.

Like Swift, and possibly for the same reason, Beethoven was unmarried; though there is ample evidence to show that he was attracted to women. I think that he, too, preferred not to share his affliction with someone else, though I also think that both he and Swift would have been happier men if they had.

As regards the cause of Beethoven's deafness, I cannot help agreeing with my friend Dr. Tremble and many others, that

the most likely cause of a progressive deafness starting in early adult life is otosclerosis.

Another point in favour of this is that as he became more deaf, he used to listen to the piano as he was playing by holding one end of a short stick between his teeth, the other end being applied to the piano top. Surely this is evidence of good bone conduction such as one would expect with otosclerosis. He also used hearing aids, though of course in those days they were non-electrical; but the mere fact that he even tried hearing aids suggests that he had a conductive deafness which, in the absence of any history or evidence of suppuration, was almost certainly due to otosclerosis.

Why then, it may be asked, did not Beethoven's deafness have any noticeable effect on his artistic output? Why was his music, if the argument is to be logical, not gloomy or even macabre?

So far as his everyday life is concerned it seems that he behaved in the conventional way of the hard-of-hearing. He was difficult, quarrelsome, suspicious and withdrawn; but his artistic output does not appear in any way to have been influenced by his deafness. His eighth symphony is as gay and light as his second, and it is difficult, nay impossible, to believe that any of his compositions could have been improved upon.

I think that one reason that his deafness did not affect him more seriously is that his was a conductive deafness. He did not have to suffer the distortion and discord of the fluctuating deafness of Meniere's Disease when the very sound of music can be intolerable; moreover, he was not like Goya, suddenly cut off from all outside sounds. His was a gradually increasing deafness which did not become profound until the pattern of musical expression was well established in his mind. After that, what was important was what he created in his mind and what he could write down; not what he heard. For him the supreme tragedy was that he could not hear what he had composed. Though he knew it was right, he did not have what must be the consummation of musical experience, the

ability to hear his own work played back to him by a full orchestra.

No doubt many other examples of the influence of mental and physical disorders can be adduced, but equally there can be no doubt that in all three patients I have mentioned, deafness was a great burden, and that in at least two of them it affected their artistic output, whilst in the third it robbed him of the fullest appreciation of his own genius.

VIIeme INTERNATIONAL CONGRESS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY.

The Seventh International Congress of Otology, Rhinology and Laryngology will be held in Paris, July 23-29, 1961, at the New Faculte of Medicine, Rue des Saints-Peres. The program includes *1.* The Recent Acquisitions, Pathology and Therapeutics of Otosclerosis by Profs. Sourdille, Sercer and Krmpotic, Weber, Ardouin, Larroude, Ferreri and Shambaugh. *2.* Indications and Five-Year Results in Surgery and Radium in the Treatment of Cancers of the Larynx and Hypopharynx—Profs. Pietrantoni and Agazzi Alonzo, Casadesus, C. L. Jackson, Leicher, Leroux-Robert and Ormerod. *3.* A Report on Allergy and Infections of the Nose and Bronchus—Profs. Van Dishoeck, Clerici, Hlavacek, Voohorst, Mayer, Laskiewicz and Terracol.

Tours and entertainment for guests and their ladies have been arranged. President: Dr. Maurice Aubry; Secretary General: Henry Guillon. For further details write Dr. Guillon, 6 Avenue McMahon, Paris, France.

AURAL CHOLESTEATOMA. EXPERIMENTAL OBSERVATIONS.*†‡

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Several theories have been proposed to explain the appearance in the middle ear of stratified squamous epithelium and subsequent formation of cholesteatoma. Clinical and histopathological evidences indicate, however, that in most cases, the squamous epithelium originates in the epidermis of tympanic membrane and/or skin of external canal. The extensive literature on this topic can be found in the publications of Nager,[1] McKenzie,[2] Lindsay,[3] Teed,[4] Day,[5] Ojala and Saxén,[6] Rüedi[7,8,9] and Friedman.[10]

Although the immigration theory has been accepted by most investigators, not all opinions agree regarding the mechanism underlying the ingrowth of epidermis. It is accepted that the epidermis may immigrate through a perforation in Shrapnell's membrane or a marginal perforation in pars tensa; however, since Nager[1] stated that "no cholesteatoma can arise with a central perforation," the possibility that the epidermis of the pars tensa can grow inwards through a central perforation has been frequently overlooked.

The controversy has been mainly about the conditions predisposing to and causing attic cholesteatoma and also whether a perforation is necessary for ingrowth of epidermis. The aural cholesteatoma following necrotizing otitis media had been disregarded since the widespread use of chemotherapy and antibiotics has reduced its incidence considerably.

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Clinical observations indicate that attic cholesteatoma may develop in two ways[11,12]: in one, there may be no history of recurrent otitis media, and Shrapnell's membrane presents a small perforation but little or no retraction. Histologic findings in experimental animals[11] suggest that this cholesteatoma may originate from epidermis which immigrated through the perforation. The other type observed clinically is preceded by repeated episodes of acute middle ear inflammation leading to formation of a retracted pocket. The development of this pocket seems to be due to cicatricial retraction of inflammatory tissue contained in the epitympanum. If a subsequent acute middle ear inflammation produces swelling, exudation, and exfoliation of epidermis with retention of debris in the pocket, then cholesteatoma may develop either with or without a perforation of the retracted Shrapnell's membrane. Further development of the cholesteatoma may sometimes be averted by prompt removal of the debris. As formerly[12] pointed out, the previous explanation applies as well to those cases of cholesteatoma which develop by way of the posterosuperior quadrant of the pars tensa. In these cases the inflammatory process destroys the membrana propria (or dense fibrous tissue layer). That part of the membrane is then devoid of its supporting middle layer. As adhesions form and later contract the unsupported part of the membrane is retracted beneath the annulus; thus the conditions for development of cholesteatoma during a subsequent acute episode are established as in the case of retracted Shrapnell's membrane.

The clinical assumption that the epidermis may ingrow in the absence of a perforation is supported by histologic studies in both man and experimental animals.[6,7,8,9] According to Ojala and Saxén,[6] Shrapnell's membrane is provided with a loose, connective tissue beneath the epidermis as a continuation from the skin of the external canal. This layer, known as the corium, prevents invasion of basal cells into Prussak's space and the attic; however, inflammatory processes may produce clefts or openings in it through which the stimulated basal cells may grow as finger-like projections. Depending upon the inflammatory conditions in Prussak's space and

attic, the invading basal cells extend over the surface of the damaged lining or underneath the mucosa or through granulation tissue. Cornification of the center of the finger-like projections of basal cells eventually may take place, thereby developing a cholesteatoma without perforation of Shrapnell's membrane. Rüedi[7,8,9] emphasized the potentialities of basal cells to proliferate and that under suitable conditions these cells may form epithelial cones which can invade the epitympanal space in the absence of perforation.

The study to be reported here will describe alterations in the middle ear of experimental animals which may have a bearing on the pathogenesis of aural cholesteatoma. The investigation illustrates the histologic picture of the epidermis invading the middle ear through a marginal perforation, through a central perforation and from an apparently intact eardrum. In addition, the questions of how the migratory epidermis progresses and the clinical implications of the findings are discussed. The observations to be reported here have no bearing on the pathogenesis of true cholesteatoma which sometimes involves the petrous pyramid, nor do they contribute evidence on the question of metaplasia of middle ear cuboidal epithelium to stratified squamous epithelium.

METHOD.

The investigation was carried out in 30 guinea pigs. The experimental procedure has been described in detail elsewhere.[13] Essentially, the method consisted of injecting 0.10-0.12 cc. of a 1.0 per cent aqueous solution of quinine hydrochloride or 0.8 per cent alcohol solution of quinine sulfate, slowly into the antrum. None of the quinea pigs showed signs of otitis media when the drug was administered.

Twenty-four animals were killed and fixed by intravital perfusion with Heidenhain-Susa solution at convenient intervals ranging from one to 76 days after the injection. The temporal bones were removed in one block and processed according to standard histologic technique for hematoxylin-eosin stain. The serial sections were studied under regular light microscopy.

Six guinea pigs were injected with quinine as described above for observation of immediate changes in the tympanic membrane. For this purpose, the external canal was cut transversely near the eardrum and observed directly under a high power dissecting microscope (40X).

RESULTS.

A. Acute Stage.

Direct observation of the eardrum revealed that it became vasoconstricted soon after the quinine solution touched the inner surface. In one hour or less, bullae appeared in the pars flaccida and/or pars tensa. The bullae enlarged slowly covering as much as half of the eardrum. The content was evacuated in a few hours into the external canal. The histologic examination showed that the bulla was formed between epidermis and membrana propria and that in the latter microscopic perforations could sometimes be observed.

On the third day, the epidermis of the eardrum showed striking hyperplasia and exfoliation. Perforation or necrosis of the tympanic membrane sometimes occurred. The lining of the tympanic cavity presented thrombosis, hemorrhages and edema in the substratum and the epithelium was necrotic in some areas.

Commentary. The formation of bullae, vascular changes and hyperplasia of the epidermis of the eardrum represented a reaction to the irritating agent. Other substances applied over the inner[8,9] or outer[14,15] surface of the eardrum have produced a similar hyperplastic reaction.

The importance of these findings is that by means of an irritating agent the epidermis of the tympanic membrane and probably also the adjacent skin of the canal were strongly stimulated, thereby setting up a favorable condition for proliferation.

B. Chronic Stage.

It is probable that the quinine was absorbed or eliminated in a few days, but the resulting inflammatory reactions or its

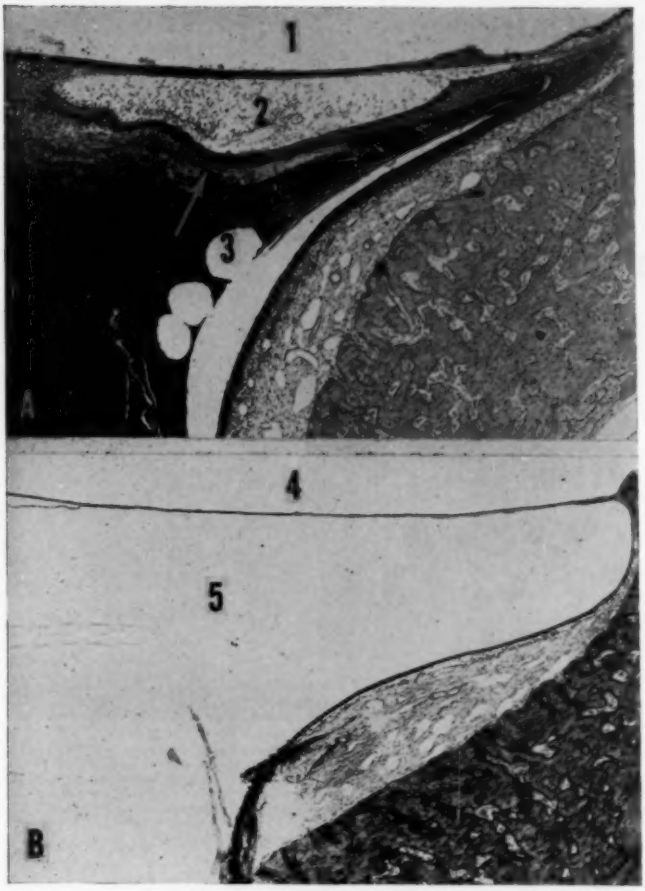

Fig. 1. Guinea pig D-50. Animal sacrificed seven days after injecting quinine hydrochloride. A.—Hyperplasia of epidermis of the eardrum. 1, tympanic cavity; 2, bulla formed by detachment of epidermis; 3, external canal filled with pus and debris. Arrow points to hyperplastic epidermis (H-E, X44). B.—Control ear. 4, tympanic cavity; 5, external canal. At the upper left corner, the epidermis was detached from membrana propria. This is an artifact. Compare its thickness with that of epidermis shown in upper photomicrograph (H-E, X44).

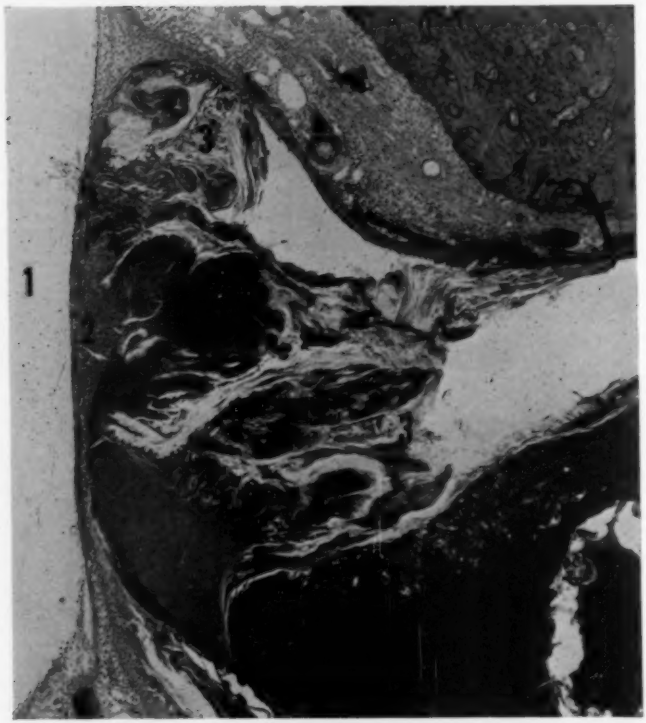

Fig. 2. Guinea pig D-50. This photomicrograph illustrates hyperplasia of the epidermis of eardrum and cholesteatoma-like formation in the external auditory canal. The animal was injected with quinine hydrochloride and sacrificed seven days later. 1, tympanic cavity; 2, epidermis of tympanic membrane; 3, debris and desquamated cell layers accumulated in the external auditory canal (H-E, X55).

sequelae could be traced in all animals. In many cases the histologic features appeared as a mixture of inflammatory reaction to the irritant and infection; however, the latter was not determined bacteriologically.

The histologic examination of these specimens showed a variety of lesions, demonstrating that the epidermis of both the skin of the canal and the tympanic membrane may immigrate into the middle ear.

I. Inflammatory Lesions.

In about seven days, the hyperplasia of the epidermis reached a maximum thickness of 10 to 15 times that of control ears (see Fig. 1).

The membrana propria did not show signs of proliferation of fibers while the epithelium of the mucosa sometimes showed hyperplasia. In some instances cornification and exfoliation of the epidermis of the eardrum produced a cholesteatoma-like formation in the external canal (see Fig. 2).

Several ears showed necrosis of the eardrum or perforations associated with otitis media. Purulent collections were found between the membrana propria and epidermis in many cases. These collections were identified as having occurred in bullae as already described.

Fibrous tissue and new bone, intermingled with bone resorption, proliferated extensively in the tympanic cavity (see Figs. 5 and 9). This proliferative reaction sometimes invaded the cochlea through both the round window and the otic capsule. Labyrinthitis ossificans, ossification of the ossicular chain and even sequestration of the cochlea were found.

Commentary. These observations indicate that irritative agents produce hyperplasia of the epidermis of the eardrum which may give rise to a cholesteatoma-like formation in the external canal. This finding and previous investigations,[6,8,9,14,15] tend to support the opinion that irritation *per se* of the epidermis of external canal and/or tympanic membrane may play an important role in the pathogenesis of aural cholesteatoma.

The overproduction of fibrous and bone tissues represents a reaction to the poison. Fibrosis and ossification have been reported in muscles after injection of quinine.[16] Secondary infection probably played an important role. The extensive work of Friedmann[17,18] demonstrated that chronic otitis media in the guinea pig may lead to osteoplastic obliteration of the ear cavities.

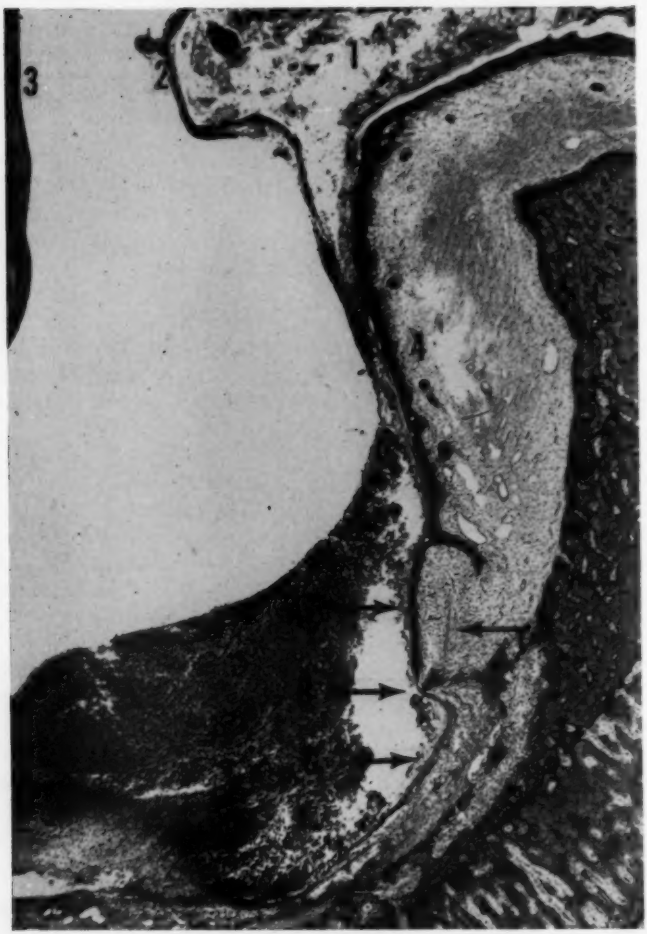

Fig. 3. Guinea pig D-31. Animal sacrificed seven days after administration of quinine sulfate. Initial stage of the migratory process of epidermis through a marginal perforation. Middle ear epithelium almost completely absent. 1, external auditory canal filled with pus; 2, malleus; 3, otic capsule; 4, skin of external canal. Arrows A, B and C point to regions of the migratory sheet which are shown at high magnification in Fig. 4. Arrow D points to remaining fibers of membrana propria. Notice continuity of epidermis of skin and migratory sheet (H-E, X31).

II. Immigration of Epidermis Through a Marginal Perforation.

The following cases substantiated the theory that the epidermis of both skin and tympanic membrane may immigrate through a marginal perforation.

Case 1. Guinea pig injected with quinine sulfate and sacrificed seven days later. The histologic examination of the temporal bone revealed necrosis of the tympanic membrane and inflammation in the middle ear. The epidermis of the skin had migrated into the tympanic cavity (see Fig. 3) where it progressed over the hyperplastic mucosa which had lost its epithelium. Arrow D points to what remains of membrana propria and arrows A, B and C to regions shown at higher magnification in Fig. 4. In A, all cells appeared slightly elongated and became progressively longer and fusiform (B and C) as the leading border of the migratory sheet was approached. The nuclei showed similar elongation and apparently did not present mitotic figures.

Commentary. This temporal bone demonstrates the initial stage of the immigration through a marginal perforation. The migratory sheet apparently presents the histologic characteristics of a repairing epithelium, *i.e.*, reproduces the skin without the corium. The photomicrographs of Fig. 4, raised the question whether progression of the epithelium is by migration and/or cell division or other mechanism. In this particular case, progression seems to be accomplished by migration of cells. The absence of mitotic figures suggests that cell division plays no part, at least during this stage of progression. A similar mechanism has been described as occurring during repair of small wounds of the cornea and skin.[16] The possibility that other processes may also contribute to locomotion of the migratory sheet cannot be ruled out. The nature of the stimulus that excites cell migration of epidermis into a region where it normally does not exist, remains unknown.

Case 2. This guinea pig was injected with quinine sulfate and sacrificed 76 days after administration of the drug. The histologic examination of the specimen showed partial obliteration of the tympanic cavity and cochlea by new bone and fibrous tissue (see Fig. 5). The eardrum and ossicular chain were destroyed, and the epidermis of the skin had immigrated and covered the surface of the remaining tympanic cavity. There had been complete healing in some areas, as shown in Fig. 5, but a study of the serial sections revealed other areas where the underlying capsule showed significant changes. In some areas, as shown in Fig. 6, the periosteum of the otic capsule had been absorbed, and the substratum of the repairing epidermis extended into the bone along blood vessel spaces and areas of osteolysis. The arrows on this photomicrograph point to microcholesteatoma formations in the epidermis. In another small area (see Fig. 7-A) a focus of bone absorption was found in the

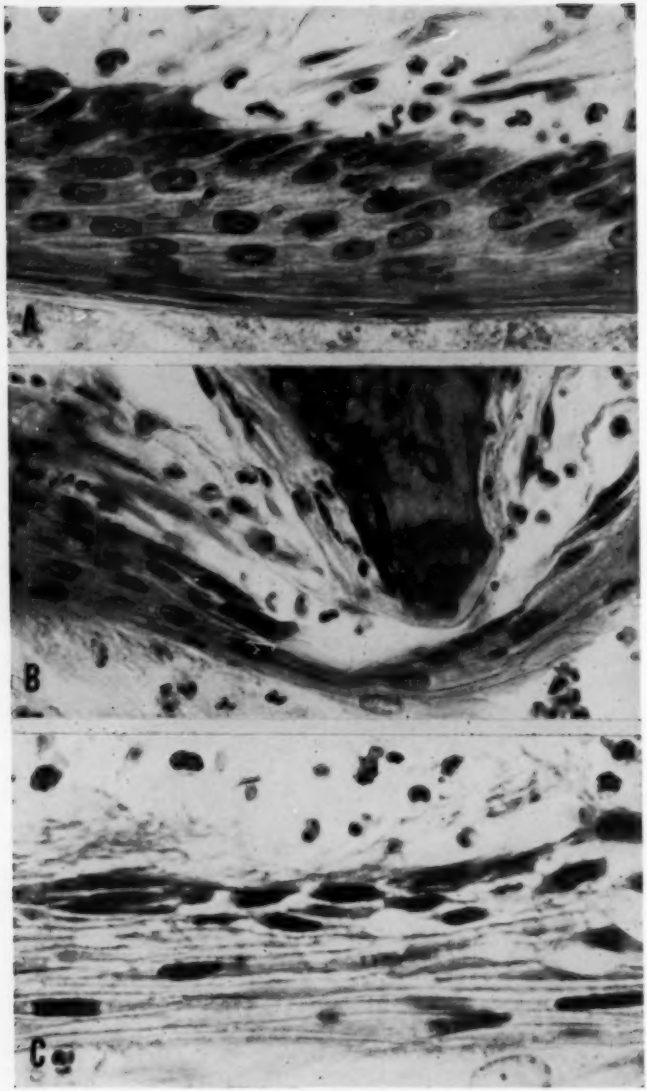

Fig. 4. Histologic features of migratory sheet in the initial stage. These photomicrographs correspond to regions pointed by arrows A, B and C, respectively, of Fig. 3. A.—Cells and nuclei are slightly elongated (H-E, X715). B.—Cells and nuclei are more elongated and mitotic figures seemed to be absent (H-E. X650). C.—Cells and nuclei are fusiform. No mitotic figures are seen (H-E, X840).

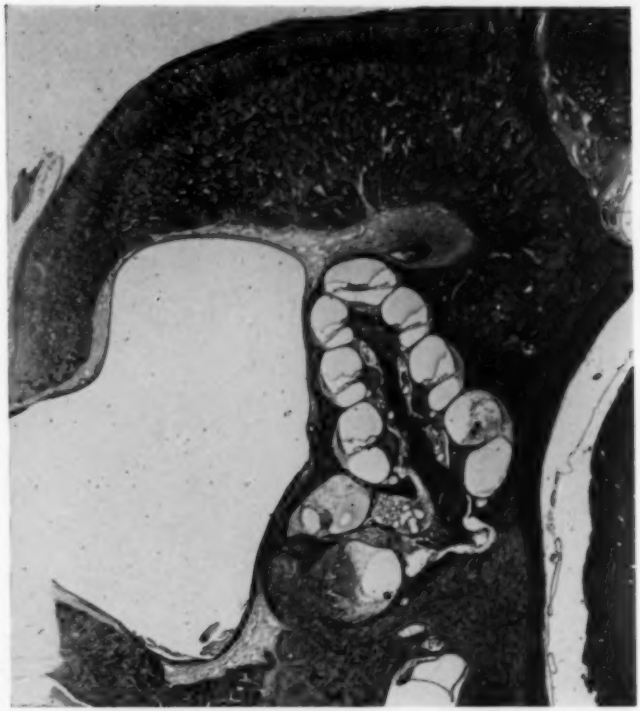

Fig. 5. Guinea pig D-43. Partial osteoplastic obliteration of the tympanic cavity and first turn of the cochlea. Eardrum, malleus and incus were destroyed. The epidermization of tympanic cavity was complete. The middle ear space appears free of infection and debris from immigrating epidermis (H-E, X13).

otic capsule under a smooth epidermis. The basal cells were more active than in other regions, and the focus of absorption presented numerous osteoclasts (large polynuclear cells in Fig. 7-B). The photomicrograph of Fig. 8 illustrated another interesting area of this specimen. It shows a small gap in the otic capsule due to a sequestrum (arrow A). The epidermis invaginated into the scala vestibuli forming a small pocket where debris accumulated in a cholesteatoma-like formation including the sequestrum. The repairing epithelium extended somewhat into the scala vestibuli (arrow B) giving a surface covering for the fibrous tissue which had obliterated the cochlear spaces.

Commentary. This case represents that final stage of the migratory epidermis. It covered completely the remaining tympanic cavity and appeared dry and smooth except for the

small region shown in Fig. 8. From the histologic point of view this repairing epidermis may be considered as the matrix of cholesteatoma.

The importance of Figs. 5, 6 and 7 is that they may have some bearing on the following question pointed out by Lindsay,[12] "In the absence of infection, moisture and retention of

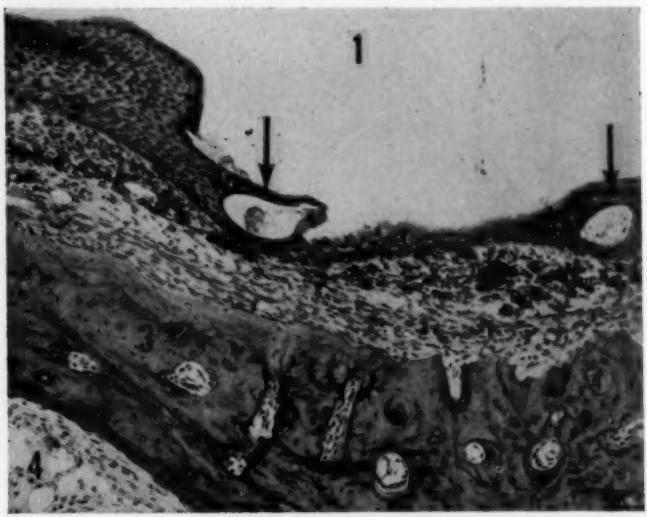

Fig. 6. Guinea pig D-43. Resorption of otic capsule under an apparently smooth repairing epithelium. The substratum invaded the bone through blood vessel channels and foci of osteolysis. Arrows point to microcholesteatomas in the migratory epidermis. 1, tympanic cavity; 2, epidermis; 3, otic capsule; 4, scala vestibuli (H-E, X130).

debris and crust *is there any evidence* that the epithelial lining derived from the cholesteatomatous matrix causes absorption of underlying bone?" This author,[12] Baron,[19] Wolff[20] and others believed that, under these conditions, there was no evidence of biological activity of the repairing epithelium. On the other hand Walsh, et al.,[21] after histopathologic studies of ossicles removed from cases of cholesteatoma and also on the basis of experimental observations, believed that the matrix

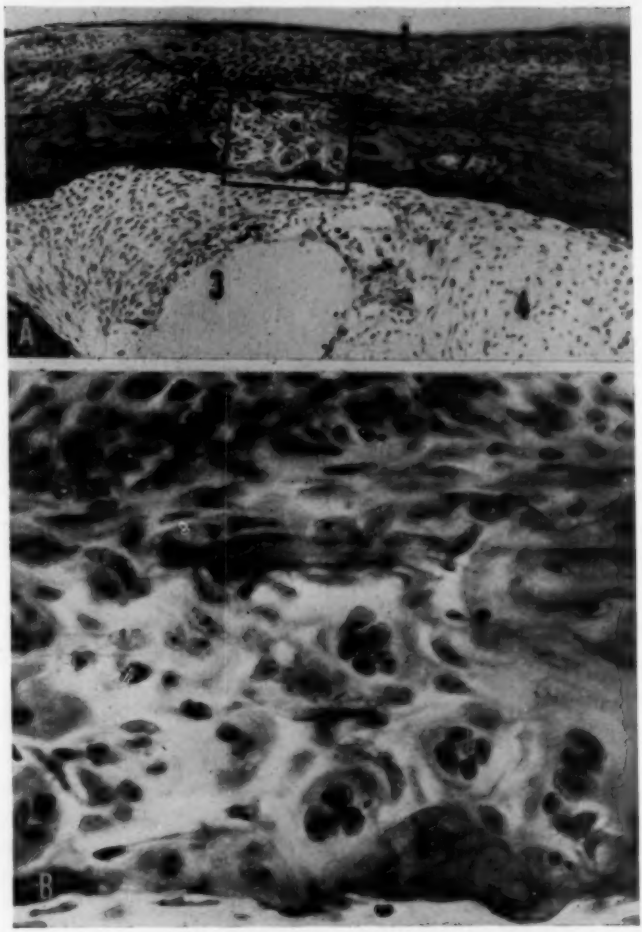

Fig. 7. Guinea pig D-43. Focus of resorption in the otic capsule underneath smooth repairing epidermis. A.—1, repairing epidermis; 2, otic capsule; 3, scala media; 4, scala vestibuli obliterated with fibrous tissue. The enclosed region is shown at higher magnification in the lower photomicrograph. Arrow points to a microscopic purulent collection enclosed in the specimen (H-E, X130). B.—Osteoclastic resorption of otic capsule. Numerous osteoclasts forming niches in the bone are seen (H-E, X330).

Fig. 8. Guinea pig D-43. Microscopic cholesteatoma-like formation on the otic capsule. 1, tympanic cavity; 3, repairing epidermic; 3, scala vestibuli filled with fibrous tissue; 4, spiral lamina; 5, scala tympani, partially obliterated with fibrous tissue; 6, new bone in scala tympani. Arrow A points to sequestrum of the otic capsule and arrow B to a transverse section of a cone of basal cells in scala vestibuli (H-E, X53).

has a biologic activity. Walsh[22] suspected that even in cases of a dry smooth cavity covered with a cholesteatoma matrix, absorption of bone may take place beneath the lining. The photomicrographs of Figs. 5, 6 and 7 might be interpreted as supporting the opinion of Walsh, et al.,[21] and Walsh[22]; however, it must be pointed out that the absorption of bone in this

instance had probably occurred during an earlier more active stage of the inflammatory reaction. The more logical interpretation is that absorption had reached its endpoint and that the reparative process was now dominant.

The photomicrograph of Fig. 8 emphasized the previous point and also that a cholesteatoma-like formation may take place in a microscopic pocket. Such a small lesion can hardly be recognized by means other than microscopic magnification.

Case 3. This guinea pig was injected with quinine sulfate and sacrificed 21 days after administration of the drug. The histologic preparations of the temporal bone showed partial obliteration of the middle ear and cochlea by new bone and inflammatory tissue. The tympanic cavity was reduced to a small pocket devoid of mucous membrane which communicated with the external ear through a narrow channel opening through a large central perforation. The hyperplastic epidermis of the eardrum immigrated through the perforation and covered the channel almost completely (see Fig. 9). The continuity of epidermis with the migratory sheet is shown in the lower photomicrograph.

Commentary. This case illustrates that under favorable conditions, the epidermis of the pars tensa has the potentiality to grow inwards similar to that of the skin. These conditions seemed to be: *1.* presence of a perforation; *2.* maintenance of a hyperplastic epidermis by inflammation and *3.* necrosis of mucosa in the tympanic cavity.

III. Immigration Without Perforation of Eardrum.

It must be clearly understood that by "perforation" is meant a discontinuity or opening which includes *all* layers of the pars flaccida and/or the pars tensa.

Clinical and histopathological evidence indicates that there are two mechanisms by which the epidermis may invade the middle ear spaces without perforating the tympanic membrane: namely, by inward displacement of Shrapnell's membrane or the pars tensa leading to a persistent retracted pocket, and by ingrowth of basal cells through clefts or openings in the corium of Shrapnell's membrane or the membrana propria of the pars tensa.

The first mechanism has been proposed several times in the past.[11,12] The progressive development of a pocket leading to cholesteatoma formation is a fact established by clinical observations. The pocket may not be perforated, at least in the

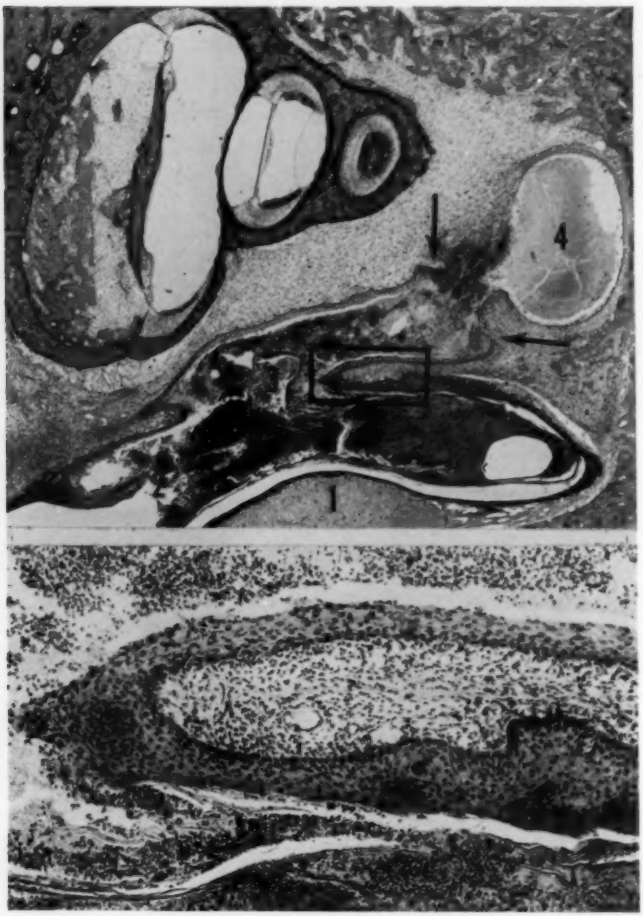

Fig. 9. Guinea pig D-46. Immigration of epidermis of eardrum through a perforation. Middle ear and scala tympani of first turn obliterated with fibrous tissue and new bone. Middle ear epithelium is absent. 1, skin of external canal; 2, external canal; 3, perforation of eardrum; 4, remaining tympanic cavity which communicates with the external ear by a channel lined with epidermis of eardrum. Arrows point to head of immigrating sheet. The enclosed region is shown at high magnification in the lower photomicrograph. This demonstrates continuity of epidermis with migratory sheet (X-120).

Fig. 10. Guinea pig D-35. Animal injected with quinine hydrochloride and sacrificed seven days later. The figure illustrates the potentialities of basal cells of eardrum's epidermis to proliferate. The hyperplasia of epidermis is striking. At this magnification (H-E, X185) a normal epidermis is barely visible. The basal cells proliferated forming long cones which extend into the hyperplastic substratum. 1, tympanic cavity; 2, malleus; 3, hyperplastic epidermis of pars tensa.

initial stages; therefore, the mechanism of this cholesteatoma is a mechanical displacement of either Shrapnell's membrane or the posterosuperior quadrant of the pars tensa. The displacement is brought about by cicatricial contraction of fibrous tissue formed in the epitympanic space during the quiescent period following an acute inflammation. The conversion of the granulation tissue to fibrous tissue and this to scar tissue

with each acute episode, is the most characteristic feature of chronic inflammation[16] wherever this may occur. Development of a pocket should be differentiated from ingrowth of epidermis; however, when perforation occurs during or after its formation then a true immigration of epidermis may take place.

The second mechanism, the ingrowth of epidermis without perforation is attributed to proliferation of basal cells. The potentialities of basal cells to proliferate are demonstrated in Fig. 10. The photomicrograph corresponds to a section passing through the malleus. The hyperplasia of epidermis and formation of cones extending in the substratum is striking.

A defect in the corium or in the membrana propria either during or without development of a pocket may permit the ingrowth of cones of basal cells in the manner postulated by Ojala and Saxén.[6] Figs. 11 and 12 illustrate the point that the basal cells may immigrate even through a microscopic opening of the membrana propria. The specimen was taken from a guinea pig injected with quinine sulfate and sacrificed seven days after administration of the drug. The lining of the middle ear was hyperplastic, and a small amount of precipitate with macrophages was found in the hypotympanum. The external canal was partially filled with debris. The epidermis of eardrum was hyperplastic and detached from membrana propria except at the umbo. The serial sections revealed two microscopic openings of the membrana propria near its attachment to the malleus (numbers 6 and 7 of Fig. 12). The basal cells of the epidermis immigrated through both perforations into the middle ear. The migratory cells presented a fusiform shape, and their nuclei were elongated.

Commentary. This case supports the opinion of Ojala and Saxén[6] and Rüedi[7,8,9] that under an inflammatory stimulus the basal cells may proliferate and immigrate without a perforation of epidermis. The microscopic opening in membrana propria may be considered as corresponding to the cleft or opening in the corium of Shrapnell's membrane, postulated by Ojala and Saxén[6]. It is apparent that these openings are not visible in the living animal because of their microscopic size and because they are covered by a hyperplastic epidermis.

Fig. 11. Guinea pig D-79. Animal sacrificed seven days after administration of quinine sulfate. The specimen illustrates the initial stage of immigration of basal cells through a microscopic defect in the membrana propria of the pars tensa. 1, tympanic cavity with a hyperplastic mucosa (arrow A); 2, bulla in pars tensa; 3, external canal containing pus and debris; 4, cochlea. Arrow B points to the region of immigration. This area is shown at higher magnification in Fig. 12.

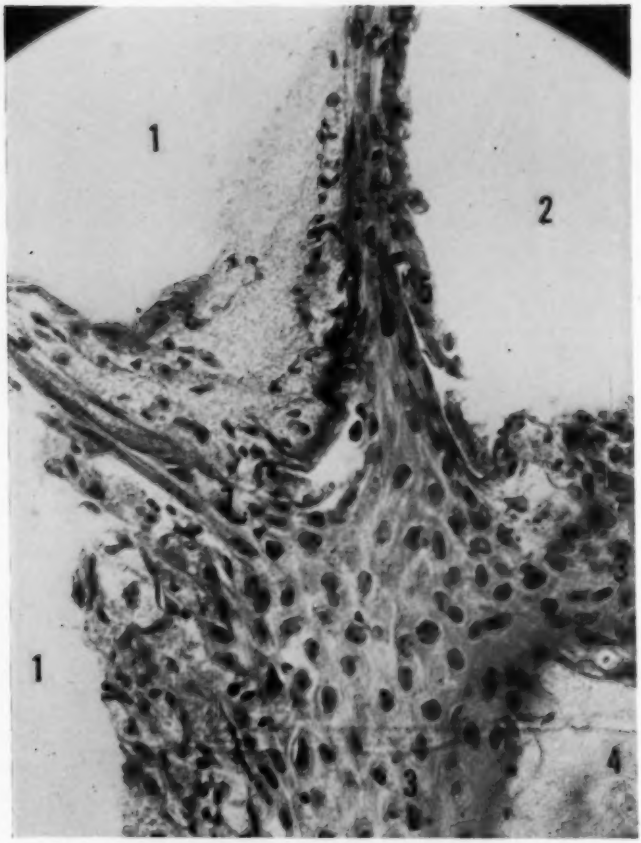

Fig. 12. Guinea pig D-79. This photomicrograph illustrates that the basal cells of epidermis of pars tensa can migrate through an opening of the mebrana propria. The epidermis was not perforated. 1, tympanic cavity; 2, bulla formed between membrana propria and epidermis; 3, epidermis of pars tensa; 4, external canal; 5, membrana propria; 6 and 7, microscopic openings in membrana propria, through which basal cells of epidermis ingrow. Notice the change in shape of migratory basal cells; the cells and nuclei become elongated (H-E, X385).

This temporal bone together with previous observations[6,7,8,9] demonstrates that the basal cells of the epidermis of a nonperforated eardrum have the potentialities to ingrow, providing the membrana propria of pars tensa and/or corium of pars flaccida are damaged. It is possible that a so-called congenital or primary cholesteatoma in the attic or middle ear may develop by this mechanism. The potentialities of basal cells to proliferate are demonstrated in Fig. 10, but the question remains whether these cones can penetrate an intact membrana propria or corium of Shrapnell's membrane.

Rüedi[8,9] observed in the guinea pig that an irritating substance applied over the inner surface of the tympanic membrane produced a strong stimulation of basal cells which migrated into the middle ear without perforation of eardrum. In the tympanic cavity a cholesteatoma developed. Rüedi[8,9] did not specify whether a defect in the membrana propria due to the inflammation had preceded the ingrowth of hyperplastic basal cells. If this was true, then the mechanism of immigration was similar to that postulated by Ojala and Saxén.[6] If there was no defect in the membrana propria, then the postulate of Rüedi implies that the basal cells can penetrate an *intact* membrana propria. Similarly, the basal cells of pars flaccida might penetrate an *intact* corium. In our series of animals no clear evidence of ingrowth of basal cells through *intact* membrana propria or corium could be demonstrated.

The statements of Ojala and Saxén[6] and the experiments by Rüedi[7,8,9] are important for understanding the pathogenesis of attic cholesteatoma, because they offer an explanation for those cases which do not show an apparent perforation of the tympanic membrane.

SUMMARY.

Histologic observations in temporal bones of a series of experimental animals exposed to the irritative effect of a suitable quinine preparation placed in the middle ear and sacrificed at various intervals demonstrated certain behavior of

the epidermis of the external canal and eardrum which is pertinent to the development of cholesteatoma.

1. The epidermis of the bordering skin can migrate through a marginal perforation when the middle ear epithelium is absent.

2. The epidermis of the eardrum can migrate through a central perforation to cover the middle ear mucosa when it is devoid of epithelium.

3. A hyperplastic basal cell layer of the epidermis can migrate inward without an apparent perforation of the eardrum providing that the membrana propia of the pars tensa or the corium of the pars flaccida has been eroded by the inflamatory process.

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HUMBOLDT UNIVERSITY OF BERLIN (CHARITE).

The celebration of the 250th Anniversary of the Charité will be held in Berlin from November 6-19, 1960, in connection with the 150th Anniversary of the Humboldt University.

Applications for participation are to be directed to the Committee for the Preparation of the 250th Anniversary of the Charité, Berlin N 4, Schumannstrabe 20-21, c/o Dozent Dr. med. habil. Dagobert Müller, secretary of the committee.

PROBLEMS AND COMPLICATIONS IN HEAD AND NECK SURGERY.*

GEORGE A. SISSON, M.D.,

Syracuse, N. Y.

The surgeon performing radical operations for the control and cure of cancer of the head and neck must accept the discouraging fact that only 35 per cent of his patients can be cured. He can prolong life in 50 per cent of his cases but do little for 15 per cent. If a surgeon is unable to adjust to these grim facts he has no business in head and neck surgery. Though his efforts may be sincere his judgment is impaired, and he will do more harm than good dabbling with something that someone else better indoctrinated might manage properly. Unfortunately, this is the first and greatest problem in head and neck surgery. Too many doctors with pessimistic attitudes are managing cancer of the head and neck in a cursory manner. What often starts as a biopsy too often ends up as an insufficient excision. This is particularly true in the management of intranasal and intraoral cancers. We continue to see nodes in the neck indiscriminately biopsied or excised when an aspiration biopsy would be a superior technique and, after all, would offer the patient a better prospect should a radical procedure be indicated in the final analysis. If the shrouds of pessimism which embrace the practice of head and neck surgery are to be removed, we, as otolaryngologists, should be the first to recognize this problem (see Figs. 1-a through 1-e).

The American Board of Otolaryngology has committeed our specialty to the responsibility of managing cancer of the head and neck. We must prove that we can accept this responsibility and actually lead the way towards improved cancer control.

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*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 8, 1960.

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Fig. 1-a. Squamous cell carcinoma of scalp in 40-year-old female; previously excised three times.

Another problem is late diagnosis. Within a three-week period I saw in consultation six cases, mostly young people, with markedly advanced lesions either at the upper limits of resectability or inoperable. In each case there was a weak link in early definitive diagnosis. One patient ignored symptoms; in another case two doctors were incapable of a complete examination of the ears, nose, throat, nasopharynx, etc.; other organs: teeth, stomach, bowel, blood stream, lungs, heart, even the psyche were thoroughly investigated while the throat and neck were overlooked. Two cases were diagnosed as cardiacs with emphysema. To the chagrin of those in attendance tracheostomies relieved both cardiacs. In another

Fig. 1-b. Lesion in old scar demonstrating extent of resection necessary to eradicate the recurrence of carcinoma shown in Fig. 1-a.

case the hypopharyngeal lesion was so massive that the top was viewed easily when using a tongue depressor. We must stress thorough examinations for minor and minimal complaints, and patients should be re-examined at intervals if the first examination is not completely satisfactory. Emphasis upon early diagnosis should be continued for lay groups as well as for professional (see Figs. 2 and 3).

Critical problems arise when selecting the borderline cases to be offered radical surgery. Almost any tumor of the head and neck regardless of its stage can be resected by the experienced surgeon; however, enthusiasm for a procedure

should not supplant mature judgment, experience or sensitive intuition. Resectability does not necessarily mean operability.

If the head and neck surgeon feels reasonably certain that surgery cannot improve, nothing should be done. A self-pitying recriminatory patient does as much to harm the cancer control program as the physician who misses an early diag-

Fig. 1-c. Same patient shown in Figs. 1-a and 1-b, with specimen showing extension of cancer into cranial wall. Cranial defect was replaced with a tantalum plate.

nosis. To be considered also are the general physical and mental qualities of the patient which might defeat successful surgery. Only once have I been advised by a competent internist that surgery was contraindicated because of cardiac reserve; however, I often have withheld surgery because of the patient's psychological background. I have seen surgery precipitate frank psychosis on two occasions; both of these patients were young females without definite aberrations in their past medical background. Conversely, I have performed

Fig. 1-d. Rotation and migration of pedicle flaps necessary to close original defect created by removal of cranial wall (see Fig. 1-b). Left lower corner shows split thickness graft used to close donor site.

radical procedures without event on highly emotional nervous individuals, young and old. In many respects the added stress placed on these latter cases seemed to recruit reserves in their personality which neither they nor their families believed present.

When the wrong case is selected for radical surgery the patient often causes others (relatives and friends) to feel that all such surgery is mutilating and thus negate its use where it could be beneficial. It has been said that there are

Fig. 1-e. Same patient shown in Figs. 1-a through 1-d as she appeared nine months postoperatively.

no contraindications to cancer surgery and this might also be said of palliative X-ray; however, unless the surgeon and radiologist agree that palliative X-ray therapy has a definitive purpose it may possibly end by being palliative only to the conscience and convenience of the doctor.

The art of treating carcinoma of the head and neck is in the physician's ability to choose the best method of therapy whether surgery, irradiation, both or neither. This ability is more difficult to acquire than the technical skill learned in the dissecting laboratory and the operating room. In neck metastasis, in carcinomas closely adhering to or invading bone, in irradiation failures and in salivary tumors, surgery usually is

Fig. 2. Far advanced inoperable carcinoma of the sinus presenting as mass in left neck.

the undisputed choice of therapy. In early laryngeal, nasopharyngeal and tonsil lesions irradiation often supercedes surgery. In most carcinomas of the head and neck combined modalities are used for best results. If irradiation is selected for the first modality the surgeon should follow the patient closely to check for recurrent disease. Conversely, if surgery is the first modality, with later use of radiotherapy likely, the radiotherapist should be invited to meet the patient as soon as possible so that he too can follow the progress and offer his modality at the propitious time. In controversial cases pretreatment conferences with both surgeon and radiotherapist in attendance are advisable.

Fig. 3. Far advanced inoperable multiple basal cell carcinoma improperly managed.

COMPLICATIONS.

The only way to avoid operative and postoperative complications in head and neck surgery is to refer these cases to someone else. The surgeon accepting total responsibility must be ready to suffer numerous disappointments and frustrations. Major complications may be classified into two categories: immediate and late. The former come from operative risks or from immediate functional changes due to loss of anatomical structures sacrificed during surgery. The

latter usually are functional disabilities and unsatisfactory rehabilitation to the loss of anatomy.

In the first group come those immediate and unexpected technical complications encountered at the operating table. Often, after several hours of dissection, a surgeon will find the lesion beyond the limits of planned surgery; this situation occurs most frequently in tongue and sinus lesions. Admittedly, head and neck surgeons deal with narrower margins of excisional safety than abdominal and thoracic surgeons. For example, one cannot always make a preoperative decision as to whether or not a commando procedure will suffice. Findings, after entering the deeper spaces, often necessitate the inclusion of the larynx and the base of the tongue. Often, to gain another two or three millimeters, it may be necessary to remove the entire tongue.

Another technical stumbling block is sudden death on the operating table. During the past eight years, with a total operating experience of over 500 cases, this has happened twice. In one case an air embolus was introduced when blood under pressure was allowed to run out. The other case, known preoperatively to be a cardiac risk, suffered cardiac arrest from myocardial infarction. We have resuscitated successfully and without incident two cases of arrest.

Pneumothorax and pneumomediastinum occur in about 5 per cent of the cases, should be suspected at once and treated by underwater drainage for three to four days. I have had two complications from "nicking" the thoracic duct and have removed "cottage cheese" from the flaps for several weeks. This is a most aggravating complication.

Sudden and extreme loss of blood can be a problem during difficult dissections of carotid body tumors or tumors adhering to the carotid bulb. These rents are repaired with arterial silk sutures and are covered with tissues from adacent areas. In one case there was a severe drop in blood pressure when a tumor was dissected from the carotid artery. This patient lived four days but never regained consciousness. We no longer graft the carotid artery; we compress it slowly over

a several-day period with the Silverstone clamp. The probability of death in using this technique is about 25 per cent. Recently Catlin, from Memorial Hospital of New York City, reported a survival after both carotids were sacrificed.

When resecting a maxilla both suctions can plug during the crucial moments. We operate with two suctions and should have three. I have sacrificed intentionally and unintentionally the seventh, ninth, tenth, twelfth and phrenic nerves. The seventh nerve is the most troublesome, but 25 per cent of these seem to regenerate spontaneously. Massive hemorrhage can complicate a procedure. Our rule is to ligate doubly all major arteries and the internal jugular. Even so, on some occasions when dissecting deep in the neck I have been compelled to remove the clavicle to stop subclavian bleeding; and when resecting massive disease at the base of the skull I have encountered massive hemorrhage from the internal jugular. In one case it was necessary to transfuse 12 pints of blood in about 20 minutes while bleeding was uncontrolled. The patient is alive today and free of disease. It has been five years since surgery.

We now close the pharyngostoma while awaiting the reconstruction phase. This procedure has spared us many immediate postoperative complications since this is certain to divert the salivary stream and prevent spit, blood and corruption from accumulating under our fresh operative flaps.

Another immediate postoperative complication is the development of aspiration pneumonia because of injury to the superior laryngeal nerve. If the brachial plexus is injured it will be detected on the first postoperative day; the patient will be unable to move his arm or fingers. This becomes a permanent disability if the nerve is resected. When the entire thyroid is removed for an infiltrating subglottic lesion we must watch for the development of tetany during the first three days postoperatively. As a precaution one can perform Sulkowitch urine examinations two or three times daily for the first two postoperative days. If there is any question of deficiency in the parathyroid hormone the usual anti-tetany measures are instituted at once. The man-

agement of postoperative fluids and electrolytes usually does not present a problem because most head and neck patients are able to be fed through a naso-gastric tube on the first postoperative day. Intake and output, however, must still be carefully checked. The pulse, blood pressure and general clinical appearance of the patient usually alerts us to severe blood loss during the immediate postoperative period. In questionable cases we use a Foley catheter for a reliable and safe check on the urine output. Applying continuous suction to wound drains has decreased the problems of postoperative hematoma. Tracheostomy is routine in commandos, bilateral neck dissections and hemilaryngectomies. With a reliable well trained house staff in attendance, should respiratory obstructions develop, one can be more liberal in partial resections of the maxilla, radical neck dissections and localized intraoral procedures.

I have seen four naso-gastric feeding tubes break away from their moorings. Once it was necessary to do a gastrostomy to remove the tube; the others passed safely through the entire intestinal tract. We now have devised a special technique for properly securing these feeding tubes.

Massive postoperative hemorrhage is always a complication. When the patient vomits a large amount of blood, and particularly if he is on the verge of shock, it is natural for the surgeon, suspecting that operative wounds are bleeding, to consider reopening the neck. This is particularly true in the more radical intraoral procedures where a suture line is exposed to the pharynx or hypopharynx. A word of caution, however. Due consideration should be given to the possibility that reopening the neck wound will not solve the problem. It is not unlikely that hemorrhage is from the gastrointestinal tract and could be a complication from the stomach rather than the head and neck. We have seen ulcers bleed under stress of surgery on two occasions.

In spite of the impressive array of antibiotics and chemotherapeutic agents which we have at hand we still must be able to recognize wound infections readily. In our experience postoperative infection usually occurs the second or third day

postoperatively, or late on the eighth or tenth day. Meticulous technique in the operating room is extremely important; two surgical masks for each assistant are mandatory and we keep conversation to a minimum. If recognized early, infection is a minor problem. We purposely withhold antibiotics prophylactically as we find it more effective to order them after the

Fig. 4. Case after total glossectomy, total laryngectomy and radical neck dissections. Wound was opened on fifth day to divert salivary stream laterally and away from the trachea. At time of photograph wound is 12 days postoperative and granulation bed is almost ready for secondary closure.

body has had an opportunity to build up antibodies during an invasive period. Often it is necessary to reopen the wound completely, divert the salivary stream and widely drain all pockets so unrecognized burrowing of infection will not continue and cause slow deterioration of the patient (see Fig. 4).

Occasionally massive sloughing of skin flaps is encountered.

This complication usually is found in cases that have had previous radiation or surgery, and it is particularly discouraging when the carotid artery is under the area which has sloughed. The surgeon can raise a skin graft on a pedicle to solve this problem; however, thrombosis of the carotid or carotid blowout with massive hemorrhage can occur at any time and cause death. We have experienced all these complications. Carotid blowout, particularly if associated with infection, recurrent tumor or postradiation change, is a hopeless situation. Lymphedema arises when both internal jugular veins have been ligated at the same time and usually occurs the second or third postoperative day, subsiding within six weeks in 85 per cent of the cases. When working around the orbit we suture the eyelids closed to prevent corneal abrasion which can be very serious. Tracheitis sicca usually occurs within the first four or five postoperative days. This occurs during the winter months and is best treated with Alevaire or Turgomist although we also use pancreatic dornase, a highly active fibrolytic enzyme.

The problem of recurrent disease is the most frustrating of all complications. If there is any question of recurrence do not temporize; the most favorable time for surgery may be lost by procrastination or deliberation. There are some people who need maximum reassurrance that all is well, and it is most difficult to tell these patients if recurrence is suspected that an aspiration biopsy, further tests, irradiation or even additional surgery may be necessary.

Salivary incontinence or drooling can be a late and disturbing complication. If all other methods fail we advocate ligation of the parotid ducts; this seems to help the situation. Unintelligible speech, dysphagia and masticatory difficulties are other long-term problems which are kept at a minimum with proper intraoral prostheses. The best protheses are those made at the time of surgery by a qualified and competent prosthodontist.

A distressing late complication is the poor adjustment of a patient to change in his appearance or disfigurement; most pronounced after major operations on the lower part of the

face. Frequently, a patient's family and friends accustom themselves to such changes, but the patient cannot. Conversely, we also find that society does not always accept these people. If this situation occurs it is tragic. When a patient adjusts to necessary disabilities it is unbelievable that the rest of us cannot. For these and other obviously related reasons we have recently organized a Cancer Rehabilitation Association in our area. We hope that this association may solve some of these problems.

SUMMARY.

Cancers of the head and neck must be diagnosed earlier and must be treated aggressively. Cancer control will advance when there are more trained otolaryngologists managing head and neck cancer. Extensive experience and insight are essential when selecting borderline cases suitable for radical surgery. Team effort among the referring doctor, the head and neck surgeon and the radiotherapist will improve control and reduce the problems and complications in head and neck surgery. The services of a competent prosthodontist must be available in communities where radical head and neck surgery is undertaken.

1200 E. Genesee St.

THE DE ROALDES AWARD.

At the recent annual meeting of the American Laryngological Association, the DeRoaldes Award and gold medal were presented to Dr. Dean M. Lierle as a great physician, surgeon, educator and humanitarian and in recognition of his services to otolaryngology and to the American Laryngological Association.

Panel Discussion on Tympanoplasty

A. C. FURSTENBERG, Moderator.

It is my pleasure and privilege to act as moderator of this Panel Discussion on Tympanoplasty. This is indeed a timely, provocative and interesting subject; it is also a complex, intricate problem which to say the least gives rise to many controversial beliefs and opinions.

The scope of this subject is infinite. New theories in the transmission of sound, new achievements in the technique of tympanoplasty, new investigations into the pathological changes in bone come to our attention almost daily. To understand them and to know their values is frustrating to say the least; however, research in this field must go on, and I have no fear that it will not, because the inquisitive impulse of otologists today is fired up and will not stop for a period of appraisal and adjustment at this stage of the program.

We are fortunate in having three panelists, each one of whom is possessed of a large and healthy curiosity, a capacity for careful observation and infinite resource. I have little hope that they will harmonize national and international discord on this subject, but I am convinced that they will make an honest, non-emotional approach to the problems of tympanoplasty, direct opinions to the real issues and give us some of the answers to the problems which need to be solved today.

I have the honor now to call upon Dr. Harold F. Schuknecht, Chief Otologist of the Henry Ford Hospital, Detroit, Mich.

COMMENTS ON TYMPANOPLASTY.*†

HAROLD F. SCHUKNECHT, M.D.,
T. MANFORD MCGEE, M.D.,
(By Invitation),
and
STANLEY OLEKSIUK, M.D.,
(By Invitation),
Detroit, Mich.

Our experience in tympanoplasty now consists of 275 operations performed during the past three years.[1] For most patients the objectives of tympanoplasty have been achieved, but for various reasons, failures have occurred in about 35 per cent of the cases. Some of these failures have been reoperated with success. It is increasingly apparent that it is not always possible to arrest the suppurative process and to reconstruct the middle ear at the same operation; it is also obvious that some failures occurred because the nature of the tympanic pathology precluded successful reconstruction.

Success in reconstructive middle ear surgery depends upon keen judgment in the selection of cases as well as the proper execution of the operation. In this regard we believe that there are several relative contraindications to tympanoplasty, the term relative applying to a combination of factors concerning the nature of the pathology and the skill and experience of the surgeon.

I. CONTRAINDICATIONS TO TYMPANOPLASTY.

A. Malfunctioning Eustachian Tube.

It is probably true that reconstructive tympanic surgery can be completely successful only when the middle ear is normally ventilated during swallowing. Often it is not possible to detect the passage of air through the Eustachian tube

*Read at the Sixty-Third Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Miami Beach, Fla., March 15, 1960.

†From the Department of Otolaryngology, Henry Ford Hospital.

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during swallowing because of swollen mucosa, secretions, polyps, etc.; therefore, we have used calibrated politzerization and have arbitrarily considered tubal function to be acceptable when air passed through with 40 mm. of mercury pressure. A tympanoplasty will ultimately fail if middle ear aeration depends upon forced inflation.

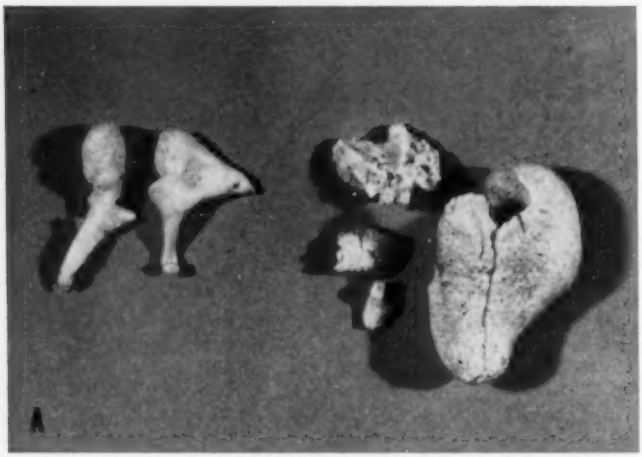

Fig. 1-a. Hyalinized collagen (tympanosclerosis) presenting as a large mass lying free in the middle ear. Normal ossicles from another patient are shown for size comparison.

B. Deficient Middle Ear Mucosa.

Examination under magnification (at least six power) is essential in preoperative evaluation for tympanoplasty, particularly for determining the status of the middle ear mucosa. The mucosal surface area must be sufficient to provide an adequate cavum-minor. Reconstructive surgery will usually fail in its objective of improving hearing if there is any destruction of mucosa in the area between the Eustachian tube and the round window. Operations to graft mucosa or to maintain the size of the cavum-minor with plastic prostheses are not yet proven to be successful.

Fig. 1-b. Hyalinized collagen surrounding devitalized crus of the stapes.

C. Hyalinized Collagen Deposits (Tympanosclerosis).

This peculiar pathological condition is characterized by the deposition of enormous amounts of hyalinized collagenous material in the submucosal layers of the tympanic cavity.[2] The ossicles may be imbedded in it (see Figs. 1-a and 1-b). Usually there are also plaques of the material within the layers of the tympanic membrane. These so-called "calcium plaques" contain insignificant amounts of calcium. The diagnosis can be made preoperatively by identification of the plaques in the tympanic membrane and grayish-white nodules in the mucosa of the promontory. These findings are particularly significant in the presence of a large bone-air gap which indicates ankylosis of the ossicles.

D. Age Over 60.

Tympanoplasty surgery does not seem indicated in patients over 60 for improvement of hearing or for elimination of discharge from a "safe" ear. Our reasons are threefold: first, there is a greater incidence of graft necroses in the older patient (probably because of vascular insufficiency in the recipient site); secondly, the incidence of pulmonary, cardiac and cerebrovascular complications from surgery are greater in the aged; and thirdly, the value received for effort is less because of shorter life expectancy.

Tympanoplasty may be performed for the healthy patient over 60 when middle ear or mastoid surgery is required to eliminate dangerous pathology.

II. CAUSES OF TYMPANIC GRAFT FAILURE.

One of the most common causes for tympanoplasty failure is loss of graft substance, a complication which may occur soon or many months after surgery. Our experiences correspond closely with those of Beickert,[3] Kley,[4] and Hohmann, et al.[5]

Full-thickness grafts from the post-auricular area were used in 219 patients between January, 1957, and October, 1959. To this date, perforations have been found in 48 (22 per cent). A second operation now has been done on 22 of the 48 failures using split-thickness skin graft. It is too early to assess the results of these revisions.

A. Early Failures (Necrosis).

Total necrosis of the free tympanic graft is rare. Partial necrosis, which is more common, results in perforation which almost never closes spontaneously. Early failure may be due to improper packing, trauma, inadequate blood supply, and infection.

There is still a difference of opinion as to whether a graft should be packed into place firmly or loosely. Certainly the principle is firmly established that the graft must be meticulously tailored to fit the contours of the recipient site, that

bleeding must be absolutely controlled, and that the graft must be approximated to its bed by packing which is firm enough to prevent collections of underlying fluid.

Neither cautery nor the polishing burr should be used on the recipient site. The polishing burr seems to plug vascular channels and devitalize bone by its heating effect. Crushing or puncturing the graft with sharp instruments causes areas of necrosis as well as possible implantation of epidermis into the deeper layers of the graft which promotes the development of epidermoid inclusion cysts and perforations.

Central graft necrosis can result when an attempt is made to bridge a large tympanic space. Ideally no part of the graft should be more than 2 or 3 mm. from a viable tissue bed; thus, spaces 4 to 6 mm. wide usually can be bridged successfully. When the entire tympanic membrane is missing and the malleus handle is missing or avascular, the central area of the graft should be placed in contact with an area of denuded promontory to ensure adequate nutrition.

Chronically discharging ears should be treated before surgery to acquire a state of remission. When suppuration cannot be completely controlled, it may be necessary to proceed with surgery even though the chances for success are somewhat less than for the "dry" ear.

B. Late Failures (Perforation).

In our experience late graft failures are caused by either circumscribed cellulitis or epidermoid inclusion cysts.

The occurrence of circumscribed cellulitis as a late graft complication is not surprising when one realizes that continuing suppuration can occur on both sides of the graft.

We have examined histologically a series of 20 tympanoplasty grafts having areas of circumscribed cellulitis and/or perforations removed six months to two years after implantation. All were full-thickness skin taken from the postauricular sulcus and thinned with sharp scissors.

The areas of circumscribed cellulitis appeared grossly as discrete, smooth, reddish areas measuring 1 to 2 mm. in size

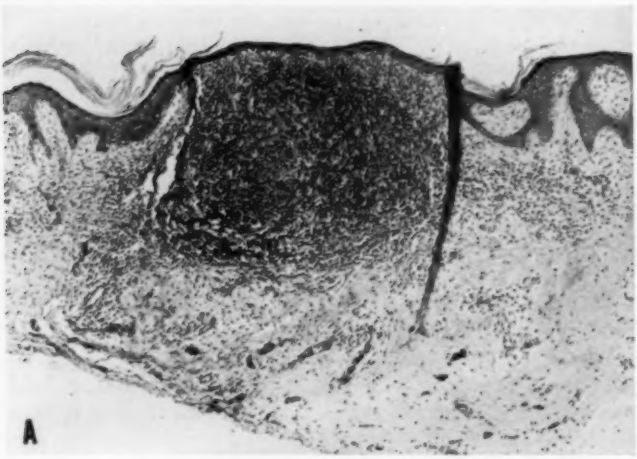

Fig. 2-a. Full thickness tympanoplasty grafts showing how perforation develops from circumscribed cellulitis. a.—Circumscribed cellulitis (full-thickness graft removed 18 months after operation).

Fig. 2-b. Cellulitis with ulceration (full-thickness graft removed four months after operation).

and were characterized by an accumulation of chronic inflammatory cells in the dermal layer (see Fig. 2). The overlying epidermis atrophies so that the rete pegs and papillae disappear. Further thinning of the epidermis results in ulceration, and finally perforation. Perforations occurring on the basis of cellulitis are often lined with mucosa.

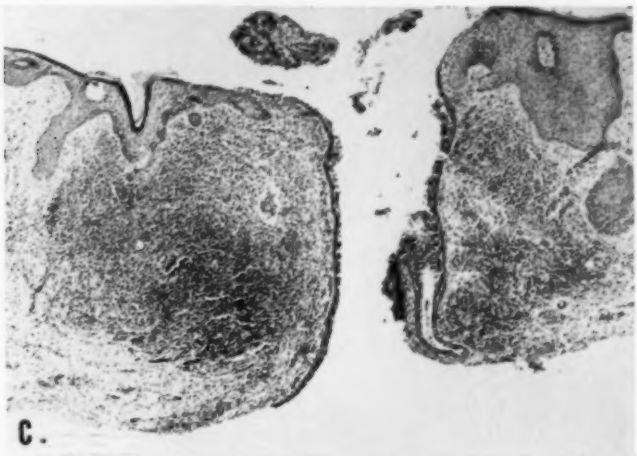

Fig. 2-c. Cellulitis and perforation with mucosal lining (full-thickness graft removed 26 months after operation).

A more common type of perforation is caused by formation of an epithelial inclusion cyst within the graft (graft cholesteatoma). A majority of these cysts probably arises from skin appendages (hair follicles and glands). In the process of thinning a graft it is possible to expose or cut the papillary ends of hair follicles which may result in perforation, or squamous epithelialization of the tympanic surface (see Fig. 3).

Other causes for inclusion cysts are infolding of epidermis due to improper positioning of the graft, and puncturing, crushing or otherwise traumatizing the graft so as to bury epidermis into its deeper layers.[6]

Fig. 3-a. Full thickness tympanoplasty grafts showing how perforation develops from epidermoid inclusion cysts. a.—Small inclusion cyst in the epidermis (full-thickness graft removed 18 months after operation).

Fig. 3-b. Inclusion cyst deep within the wall of the full-thickness graft. (Removed 18 months after operation.)

We have observed the occurrence of inclusion cysts two years after an initially successful operation.

The mucosal lining on the tympanic surface of the grafts consists predominantly of a fragile transitional-like epithelium which is two or three cells in thickness. In the anterior mesotympanum the lining is sometimes of the pseudostratified columnar ciliated type (see Fig. 4).

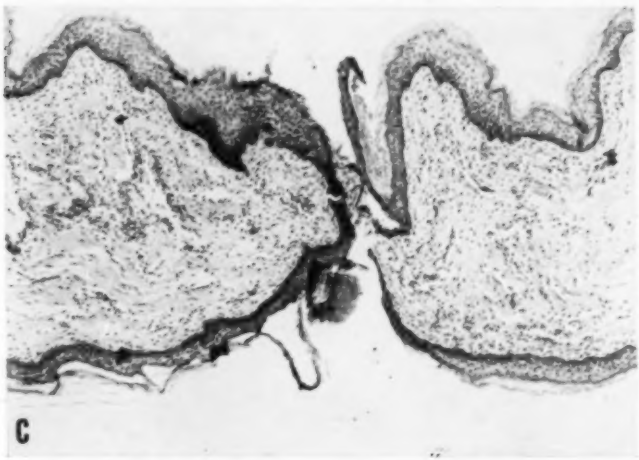

Fig. 3-c. Small perforation with squamous epithelial lining (removed 11 months after operation).

C. Current Grafting Methods.

For defects of the membrana propria which involve up to 25 per cent of the total area we now use the vein graft according to the method of Shea.[7] For defects involving from 25 per cent to 75 per cent of the membrana propria we follow the suggestion of Dr. William House,[8] using full-thickness skin from the anterior wall of the auditory canal (also advocated by Plester[9]). This appears to be excellent graft tissue because it is free of hair and contains few glands. These skin grafts are not large enough ordinarily to close complete drum de-

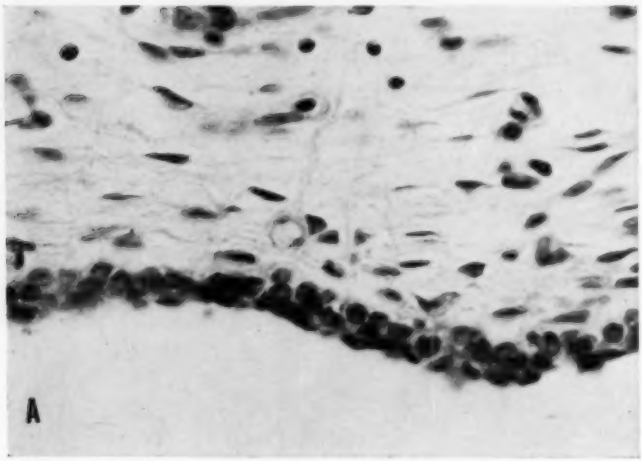

Fig. 4-a. Mucosal regeneration on tympanic surface of grafts. a.—Transitional-cell type epithelium on graft removed 18 months after surgery.

Fig. 4-b. Pseudostratified columnar ciliated epithelium on graft removed five months after surgery.

fects. For reconstructions requiring larger grafts, we now use split-thickness skin (.018 inch) taken from an area with the least hair. The donor site is selected from the arm, chest, or thigh with the aid of magnification.

CONCLUSIONS.

Reconstructive middle ear surgery is usually unsuccessful

Fig. 4-c. The mucosal lining of tympanoplasty grafts is very fragile and can be lost in preparation. The absence of a mucosal lining on histological sections should not be misinterpreted to mean it was not originally present.

in the presence of the Eustachian tube malfunction, hyalinized collagen deposits, and deficient mucous membrane; ordinarily it should not be performed for patients over 60 years of age.

Skin graft failures can occur immediately, or many months after surgery. Graft failures can be decreased by the proper selection of the donor site, careful handling and approximation of the graft to the recipient site, use of cutting burrs only, and by controlling suppuration.

Tympanoplasty will achieve and maintain a position of

respect among otological procedures only if we, as surgeons, properly assess the underlying surgical pathology and achieve appropriate microsurgical skills.

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DR. FURSTENBERG: I now have the privilege and the honor of calling upon Dr. Joseph A. Sullivan.

TYMPANOPLASTY.*†

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Accurate foreknowledge elevates the art of medicine into a science and forges its diverse elements into a glowing link between the immortal achievements of the past and the infinite prospects of the future.

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The true physician is no soothsayer, but rather one who opens the curtains and windows of the sick room to let in the light of wisdom and the fresh air of common sense.

Authorities who have given the most attention to prognosis dwell perpetually upon decorum and dignity. We learn from their works that the establishment of a proper regime, abandonment of past errors and expectant treatment are the altars upon which "recent advances" must be laid and from which all will soon be discarded that is of no permanent value.

In surgery, the decision to operate is still comparable with an act of war, though many operations formerly attended by high mortality may now be undertaken in safety: having been trained primarily in general surgery, experience teaches how, at the onset of an operation, to identify vulnerable nerves and to ligate arteries which supply redundant tissues, thereby preserving them. The simplicity of an operation has always been the measure of its perfection. The unexpected and the uncomprehended are our main adversaries.

If, from time to time, we are—so to speak—taken into the confidence of the Almighty, in the exercise of our profession, we should be careful how we impart our knowledge to the patient. Humanity must be observed and preserved.

The attitude of otolaryngologists to our specialty should be one of gratitude that antibiotics have made safer the many new procedures in this field which are now so commonplace.

The essayist, in a paper read before the Canadian Otolaryngological Society, June, 1957, had this to say: "Many of us might consider that we are living in one of the most progressive eras of our specialty, but let us approach any medical subject with humility; for, knowledge is immense, and the spirit of man can extend infinitely to enrich itself daily with new requirements. Many of the accepted facts of today may in time become the errors of yesterday. Especially is this true of the function of hearing, and the 'resurrection' of certain so-called new procedures within the middle ear."

The hopes of a great many deafened people have been

raised by the development in recent years of three operative procedures for certain types of middle ear deafness.[1]

1. Fenestration.

2. Stapes mobilization.

3. Tympanoplasty.

In another paper written over 15 years ago, the author commented as follows:[2]

"The treatment of the chronically discharging middle ear depends on an understanding of the histological and physiological problems concerned. It is our constant endeavor with our present knowledge and the application of the newer antibiotics in otologic treatment to prevent a radical procedure if at all possible. The successul interpretation and treatment of chronic middle ear disease requires a clear understanding in each case of the etiology and of the pathology responsible for the chronicity. The aims of treatment are:

1. To eliminate the danger of intracranial complications.

2. To obtain a dry ear.

3. To preserve, and if possible, to improve the hearing."

I went on further to say: "Some form of meticulous surgery with magnification in the middle ear will give the best chance of preserving hearing, but unfortunately the hearing picture in mastoid surgery for many years seems to have been of secondary importance. Great care is exercised in removing granulations and diseased tissue from the area of the oval and round windows. This surgery is followed by placing skin grafts in the middle ear on a mobile stapes and in the mastoid cavity. If granulations can be prevented from forming in the above areas and subsequent scarring avoided, it is hoped that the round and oval windows will continue to function and the hearing be maintained or improved."

I mention one type of chronic otitis purposely, namely the allergic type.[3] Allergy is a term often used to cover ignorance or give a touch of mystification to the process, yet many

destructive lesions are in reality a hypersensitiveness of the tissue to bacterial proteins.

One should remember that redness of the presenting tissue does not exclude allergy, and this process also explains in part the active response of tissue to an acute exacerbation in a chronic ear. The initial lesion in the body may be insignificant and far removed, carried by the blood stream and producing an ID reaction in the ear. Control of the nasal condition by discovery and elimination of the allergy factors may result in a cessation of the aural discharge, and the perforation may then be closed.

In 1938 Lempert described the one-stage fenestration operation with the viable tympano meatal flap which he had previously used to seal a labyrinthine fistula in the performance of an endaural mastoidatticotomy for chronic epitympanic suppuration. This was one of the greatest advancements in temporal bone surgery and from it numerous modifications evolved.

Following this successful fenestration surgery, otologists again became aware of surgery as a means to improve hearing. From that period on, numerous articles appeared in the literature on the Bondy type conservative mastoidectomy; thus the preservation of hearing in surgery for chronic ear disease was made possible by the techniques, the magnification and the illumination which were originally employed in the operative treatment of otosclerosis.

With the invaluable aid of chemotherapeutic and antibiotic drugs, associated with a better understanding of skin grafting techniques and increased knowledge of the physiology and pathology of the ear, otologists now strive to eradicate the pathology and maintain or restore the function of the diseased middle ear. In other words, the essence of these new techniques is the direct application of the fundamentals of adequate temporal bone surgery superimposed with the principles of plastic surgery to otology.

Otologists for years have been aware of the necessity for reconstructive surgery in chronic ear disease. We have a

number of patients operated by the complete radical procedure who have maintained or gained a useful level of hearing. Following various forms of modified radical procedures we have had numerous excellent results in maintaining or restoring hearing; however, these results were obtained without a clear understanding of middle ear physiology. We failed to apply the principles of the middle ear sound pressure transformer which was clearly described by Helmholtz in 1863. Finally it remained for Zollner[4] in 1951 and Wullstein[5] in 1952 to apply the mechanical principles of the middle ear transformer to the surgical reconstruction of the middle ear following destructive middle ear disease.

Chronic otitis media causes pathological and functional defects of varying degrees, hence the method to restore the hearing must vary. Stacke, writing in 1897 said, "No one will bind himself to one method in operating; rather will every thoughtful operator individualize and modify the method from case to case according to the particular pathological condition."

All these variations in technique, however, must be governed primarily by striving to eradicate the disease completely and second, to establishing a sound conducting apparatus.

What do we mean by the terminology "tympanoplasty"? Well, it does not mean what it is supposed to mean, namely—a combination of surgery for eradication of chronic ear inflammation and surgical reconstruction of the sound conducting and sound protecting mechanism. Rarely can it accomplish both of these requisites at one operation.

We owe a great debt to Wullstein, who has classified reconstruction of the sound conducting apparatus into five types.

Type I—Rebuilds a normal ear—tympanic membrane with both functions: sound protection for the round window and the scala tympani, and sound pressure tranformation over the ossicular chain to the scala vestibuli.

Type II—Tries to preserve, in spite of slight defects of the

ossicles, a middle ear of about normal size and depth between tympanic membrane and promontory.

Type III—In this type, large defects of the malleus and incus make the removal of the ossicular chain and the epitympanum necessary. The tympanic membrane, in order to function as a sound pressure transformer, must be directly connected with the head of the stapes. This has been called, Columella-Effect by Zollner and Myringostapediopexy by Juers. This was originally done by Matte in 1901. This is a comparable problem that we are often called upon to deal with in the most difficult of all otologic surgical procedures, the repair of the congenitally atresic external ear canal and maldeveloped mastoid bone and middle ear remnants.[6]

Type IV—In this type, the stapedial footplate is movable, but the crura are missing. It is impossible to obtain sound pressure transformation. The oval window is left open to the auditory meatus. The tympanoplasty reconstructs a middle ear consisting only of the tube and hypotympanum with wound protection for the round window.

Type V—In this type, the stapedial footplate is fixed, and a fenestration of the horizontal canal is required; however, it is nccessary to protect the round window.

The post auricular incision is preferred by some physicians, particularly when a post auricular skin graft is employed. The post auricular graft can be obtained from the same incision; we prefer the skin of the forearm or the anterior canal wall as I will describe later on.

I believe that the results in many cases do not justify this tedious meticulous surgery, because the hearing improvement is not predictable. It is true the ears can be tested to determine the cochlea function, but you cannot test in an accurate manner how the middle ear will function after the procedure is completed. In simple perforations with the ossicular chain intact wherein a paper patch restores hearing to a high degree, then of course, one could expect a similar result by tympanoplasty; but the hearing must be good to begin with to obtain any kind of satisfactory result. True, once in a

while you may get a spectacular result, but this type of improvement cannot be obtained constantly.

I am for any kind of meticulous surgery which cleans the disease from the most remote areas heretofore neglected. As one who has used the dissecting microscope, not just a loupe, from my early work in the facial nerve, beginning nearly 30 years ago until the present day, I am fully cognizant of this fundamental prerequisite. Bekesy, Wever and Lawrence have clarified and established the psychoacoustic principles which are involved in obtaining an air space in the region of the round window. If the stapes is not movable then, of course, creating an air space in the round window is useless. Then comes the question of whether to fenestrate if the stapes is fixed. Logic postulates that if an artificial membrane can be created in the middle ear with an air space in the region of the round window and a mobile membrane created over the fenestra, then there should be some improvement of hearing.

My associate, Dr. Kenneth McAskile,[7] in a paper read before the Canadian Otolaryngological Society in 1959, reported a method of repairing a large marginal tympanic perforation using a pedicle flap which can be rotated either from the floor or from the anterior wall of the external canal.

It is well known that the pedicle flaps have been used for years to close defects in tympanic membranes and that at the present time most authors prefer free skin grafts to pedicle flaps because of the ease of accurate placement and exact adaptation to this defect; yet as with all skin grafts, the results of the operation should be assessed only after the lapse of many months.

Experience has shown us, however, that the tympanomeatal flap as used in the fenestration operation will endure infection and trauma while free skin grafts used to cover raw areas in the same cavity will break down or slough out completely, sometimes several years after the original operation.

In modified radical mastoidectomies with epitympanic perforations or with small perforations in the pars tensa it is relatively easy to obliterate the defect by freshening the edges

of the perforation and then approximating the margins. It is sometimes necessary to cut down through the tympanomeatal flap to the perforation in order to obtain good approximation. In these cases, as in the fenestration, the tympanomeatal flap is usually rotated in an anti-clockwise direction, the skin from the anterior canal wall being used to obliterate the epitympanic space.

In closing a large marginal defect with a tympanomeatal flap a larger portion of the skin of the anterior wall is in-

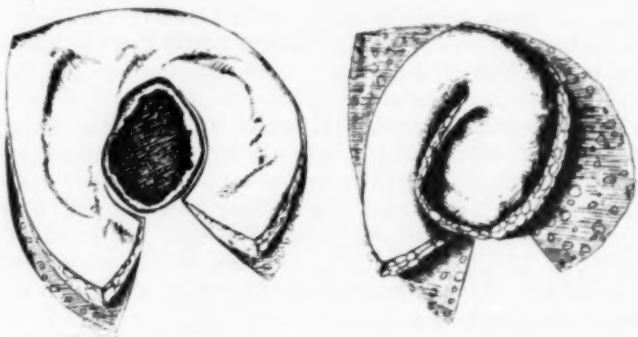

FIG. a FIG. b

Fig. 1.

cluded down to the tympanic annulus and more anteroinferiorly than is usual. A bed for the pedicle flap is prepared similar to that used in a myringoplasty or tympanoplasty by denuding the epithelium from the remnants of the tympanic membrane and the adjacent canal wall. The tympanomeatal flap is now rotated in a clock-wise direction, and the skin of the anterior canal wall easily closes the tympanic defect. The flap is then held in position by shaped cellulose sponge impregnated with an antibiotic solution or ointment. This procedure has the distinct advantage over a free skin graft in that it has an adequate blood supply of its own and also that it is tissue native to the area concerned (see Figs. 1-a and 1-b).

We have used this procedure in eight cases of total perforation and in only one case is there a remaining perforation. Unfortunately the perforation in this case is located over the round window which is not compatible with good hearing. A small piece of gelfoam inserted in this defect restores the hearing to the 20 decibel level. Conservative attempts to close this perforation have failed, so it will be necessary to use a free skin graft for closure.

The use of artificial prostheses in the middle ear was advocated by Wullstein and Zollner to replace the crura of the stapes where a mobile footplate existed; however, tissue reaction to the acrylic and vinyl plastics forced them to abandon this material.

House, Schuknecht and Shambaugh have reported the employment of either polyethylene tubing or tantalum wire to replace diseased or absent components of the conducting apparatus in the middle ear. As yet they have not noticed any local reaction to these materials. We have had occasion to use either tantalum wire or polyethylene tubing on a number of occasions and our observations have been similar to those stated above, not good.

1. Reconstructive aural surgery as a term may suggest a certain uniformity in the operation; it is different in every case, not only in details but even in the main lines.

2. We have learned that in cases of a disrupted ossicular chain, our results are much better, insofar as getting a dry ear is concerned, and also regarding final hearing, when we take away the mutilated incus and malleus and put the drum or a skin graft pushed in upon the head of the stapes; naturally, the bridge is removed. In other words when the ossicular chain is not intact, we do not try to put malformed ossicles together again. We prefer the apposition of the drum (with or without a graft to enlarge the effective size of the membrane) upon the head of the stapes. Sometimes amniotic membrane can be placed under the drum, to prevent its adherence to the promontory.

We believe that in unilateral inflammation, fenestration should be avoided.

We believe in the two-stage operation because the most difficult problem is to heal the original disease.

Lindsay states that operations in chronic middle ear infections present a more serious problem than fenestration. Individual adaptation of the operation and diagnosis require great versatility, and the final result places heavy demands on the patience and endurance of surgeon and patient.

To summarize:

1. It is impossible, without exploration of the middle ear under magnification to make a prognosis as to functional improvement that may be gained by a tympanoplasty. If there is gross scarring of the window regions an improvement of function is unlikely to take place.

If the disease is minimal the defect in the drum is repaired with a full thickness graft; if it is necessary to remove the bridge a good functional result may occur in some cases providing that the incus is left with its covering of mucosa.

2. If there is gross mastoid disease with interruption of the ossicular chain:

a. A most meticulous operative toilet with removal of all disease is first carried out under magnification in all cases.

b. Adequate toilet of the window regions is carried out.

c. The tympanic reconstruction is not performed until one has an ear that is fit for it. In most cases it is not performed until some weeks later.

d. In regards to the mastoid cavity, the post auricular flap technique of Hynes and Beales[8] is a distinct contribution.

e. There must be a functioning Eustachian tube.

McAskile in closing his paper states—in reviewing numerous articles on this type of surgery it is interesting to note that the majority of authors put great emphasis on the func-

tional results that have been obtained but very little time or space is devoted to reporting the presence or absence of discharge from the operated ears. The main danger in tympanoplasty comes from burying infection of cholesteatoma under the skin. The primary concern in chronic ear surgery is complete eradication of every bit of pathology. If this principle is sacrificed in the hope of saving or restoring hearing we have failed in our duty to the patient.

In conclusion, the real goal is prevention: to attain this objective, we must increase our basic knowledge of hearing difficulty and be able to diagnose accurately reversible and irreversible pathology of the middle ear. Meanwhile, these plastic procedures on the middle ear are reclaiming enough hearing to give real hope for the future.

"There are many arts among men, the knowledge of which is acquired bit by bit by experience that causes our lives to move forward by the skill we acquire, while want of experience subjects us to the effects of chance."

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DR. FURSTENBERG: It is my pleasure now to call upon a distinguished guest who because of his significant contributions to otology is worthy of our highest praise. Dr. Nils G. Richtner of Stockholm.

RECONSTRUCTIVE MICRO SURGERY OF THE EAR, ESPECIALLY WITH THE CAVUM MINOR TECHNIQUE.*†

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In the last 20 years there has been great progress in surgery of the ear, resulting especially from the introduction of the microscope in ear surgery. Its use was initiated by the Swedish professor Nylén and subsequently carried on by Gunnar Holmgren from 1930-1945, who was particularly interested in surgery of otosclerosis. In this branch of surgery we have no infection of the ear. In contrast we have the chronic otitis giving rise to impaired hearing. For many years this has also presented a great problem to all ear surgeons leading to new operations in an effort to determine what will produce the best results in hearing and healing. Earlier one operated primarily to obtain a dry ear and gave little thought to the patient's hearing; but since the inception of the audiometric era, starting with Fowler, Sr., the present aim of surgery is to obtain not only dry, healed ears after operation but also to give the patient better hearing.

Plastic surgery of the middle ear has gained tremendous ground in otology especially through the outstanding work of Wullstein and Zöllner. The patient with chronic middle ear inflammation, frequently with greatly impaired hearing, can now be relieved of his infection, of the false tumors or so called cholesteatomas frequently accompanying these forms of ear inflammation, and also enjoy a certain degree of improvement in his hearing.

Thanks to the advances made by these eminent workers in otologic surgery this has become possible not only through radical operation on the cellular system of the ear as per-

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formed earlier but also through plastic operation on the elements of the middle ear, the mucosa, the auditory ossicles and the tympanum, resulting, in many cases, in astonishing improvement of the hearing. The operation of chronic otitis and mastoiditis with reconstruction of the ossicles and covering of the drum with a skin graft is called tympanoplastic surgery.

Sometimes we also have the so-called myringoplastic surgery in which an attempt is made at plastic closure of a greater or lesser perforation of the tympanon peraurally without disturbance of the ossicles. The indications for this approach are limited, however, and it must be done with great caution. The perforation of the drum must be centrally situated and must not involve the limbus of the tympanic membrane in any way. The ear shall have been dry for several months and no X-ray changes shall be present in the cellular system of the middle ear indicating inflammation there. The preoperative audiologic test, the transmission investigation by means of a vibrating probe and the prosthesis test shall give definite evidence of satisfactory function in the ossicle chain, and the prosthesis test must show appreciable improvement in the hearing. In cases of small perforations with a dry ear we can obtain good results, but in cases presenting large perforations the results are generally not so satisfactory.

As a transplant a meatal flap from the inner part of the external canal can be used or a free, full-thickness skin graft can be taken from behind the ear or, as has been done in some cases, mucosa from the mouth (Hall) or a vein graft as used by Shea, Jr., may be employed.

The rest of the plastic operations of the middle ear are included under the common name of tympanoplasty, and include the radical approach, either peraurally or retro-auricularly, with operation on the cellular system of the ear and creation of different types of middle ear cavities *ad modum* Wullstein and Zöllner. These operations are suitable for all forms of chronic middle ear inflammation and mastoiditis, and may be performed in a single-stage operation or, if the

inflammation is very extensive and acute, in two stages. Some surgeons do not undertake this operation until the ear is completely dry for many months; others report that they operate on patients with very severe infections and do the tympanoplasty in the same stage or a few months after the radical operation cavity has been cleaned.

There have been many discussions during recent years concerning what should be done about the large radical operation cavity. There are many views on the healing, and many surgeons contend that they do not obtain as good healing of the free full-thickness skin graft or split skin graft placed over the radical cavity. They state that skin placed over the bone does not heal and grow as well as in the tympanic cavity where there is an abundance of vessels. Sometimes the ear does not heal for many months after the radical operation, and it is very difficult for the ear surgeon to treat these patients; therefore some surgeons have discussed and in some cases have also tried to fill the operation cavity with a pedicle flap from muscles (Guilford and Rambo). Others prefer a pedicle skin flap taken from behind the ear and placed down in the operation cavity, which gives much better results and good healing after one or two months (Beales). Beales says that with a full-thickness skin graft not all cavities healed after six months, *i.e.*, not more than 30-40 per cent, while with the pedicle flap of skin behind the ear he obtains much better results. These series are not yet extensive enough to permit discussion of the percentage.

An effort must be made to close the operation cavity primarily, and I like to use the Guilford and Rambo technique to fill the operation cavity and to cover the horizontal semicircular canal with muscle. When the ear in these cases heals and the perforation has also healed with a skin graft, the ear is wholly normal and the patient is able to swim, for instance, without any danger to the ear.

Earlier I used a free full-thickness skin graft taken from behind the auricle in the same incision as the retroauricular one, but free skin grafts placed in the mastoid cavity and in the middle ear sometimes slough due to the lack of blood

supply; therefore some time ago I started working with combined skin and muscle pedicle flaps and also with the retroauricular incision, and I shall see if this method will allow the radical cavity to heal in a shorter time and maintain the flap in good condition. The pedicle flap will cover the middle ear cavity and will give a larger middle ear cavity; the combined skin-muscle flap will fill a great part of the mastoid cavity thus giving us a small meatus down to the new drum and very little cavity and in some cases no cavity

Fig. 1.

at all. This operation is especially recommended for chronic otitis with no suppuration, chronic otitis with a cholesteatoma, etc. In the presence of suppurating chronical otitis I prefer first to do the radical operation and some weeks later, when the infection is gone, to perform the plastic operation. My material is still too limited to allow any definite conclusions about the healing and hearing after this new pedicle flap operation, but it seems to be a good method and in the cases I have operated the hearing is good. I will show you some drawings from this form of tympanoplasty. Fig. 1 shows

the incision and the skin flap and skin-muscle flap. We then do the radical operation in the conventional manner, radical operation or a modified radical operation where the bridge is left *in situ*. With the pedicle skin flap it is then easy to make a cavum major or a tympanostapediopexy or a small cavity as shown in Figs. 2 and 3 and then to fill the radical cavity with the skin-muscle flap. A part of the pedicle flap must then be excoriated as you see in Fig. 3, and the posterior part of the meatus sewn to periosteum behind the skin-muscle flap.

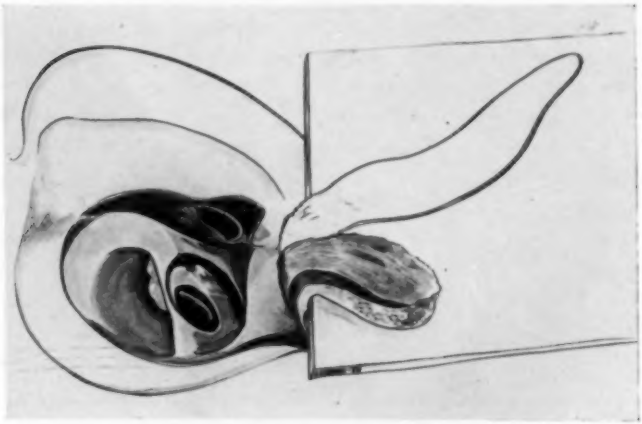

Fig. 2.

At Sabbatsberg Hospital, Stockholm, all these operations are performed under general narcosis (anesthesia with Pentotal and intratracheal intubation). A disadvantage of this procedure is that we can not test the patient's hearing during the operation, but it is very difficult for the patient to lie on the operating table the two or three hours or more that the operation requires.

All these ear operations are preceded by a clinical study of the middle ear, frequently under microscope, for evaluation of the appearance and the type of perforation and investigation of the auditory ossicles. Special measurements of the

hearing are always carried out primarily with an ordinary pure tone audiogram and sometimes complemented with a speech audiogram. The so-called transmission investigations are included in these studies; especially important are the vibrating probe and the prosthesis test, both the so-called complete prosthesis and the covering prosthesis tests. The latter involves primarily a screening prosthesis test over the round window.

Fig. 3.

In chronic middle ear inflammation where we have chronic mastoiditis, osteitis or cholesteatoma of lesser extent, that can be removed without injuring the auditory ossicles or tympanic membrane, we do the *cavum major*, the large cavity.

In a case presenting minor destruction of only the long process of the incus we try transposition of the incus so that

the long process comes into contact with the stapes. In some cases one can fix the lenticular process of the incus to the stapes with an extremely fine steel wire or with catgut, but we have seen that merely placing the lenticular process in contact with the head of the stapes resulted after some weeks in formation of fibrous tissues giving a good contact.

Transposition can also be done between the malleus, the long process of malleus, and to the stapes directly as with a columella from the long process of incus down to the stapes as has been shown by Shambaugh. We can use a small polyethylene tube on the long process of the incus and down to the stapes; in fact, many variations can be used, and it is impossible before a tympanoplastic operation to know exactly what is to be done. The decision must be made during the operation.

Sometimes we use type III *ad modum* Wullstein, the so called tympanostapediopexy, or, according to German nomenclature, the formation of "eine flache Pauke." The primary result of this type of operation has been good, with respect to healing as well as to hearing, but the late results have been less satisfactory in some cases due frequently to the occurrence of a perforation in the flap over the stapes.

In the cases presenting a pronounced defect in the ossicle chain I have employed Wullstein Type IV, the so-called "eine kleine Pauke" or *cavum minor* technique. In many cases this has afforded astonishingly good results even in cases of extremely grave auditory impairment. Many encouraging results were obtained with this technique even in the early days, five years ago, when I started seriously to engage in tympanoplastic surgery. Stimulated by the excellent results I have, therefore, been particularly interested in undertaking treatment of the extremely grave cases of auditory impairment in chronic otitis in order to see what results could be obtained with this special technique. About 200 tympanoplastic operations have been performed with not less than about 80 patients operated on according to the *cavum minor* technique, with minor variations from case to case, but with the consistent purpose of establishing a difference in pressure be-

tween the round and oval windows; consequently it is this type of operation I should like to discuss in detail now, particularly with respect to technique and the auditory results, which are frequently so good that it seems difficult to explain them from physiologic point of view.

It is true in this case as in all other plastic operations on

Fig. 4.

the middle ear that the Eustachian tube should be patent, permitting easy passage of insufflated air. The operative procedure is largely as described by Wullstein and Zöllner. First the radical operation is performed with thorough removal of polyps and granulations about the residual stapes or the footplate of the stapes and the round window and the tube without damaging the epithelium more than necessary.

An incision is then made (see Fig. 4) in the mucous membrane of the medial wall of the middle ear below the prominence of the facial nerve canal and forward over the Eustachian tube above the facial prominence so that a narrow flap is obtained there to fold up against the tube. The rest of the mucous membrane is turned downward, the epithelium forming a bed for the transplant.

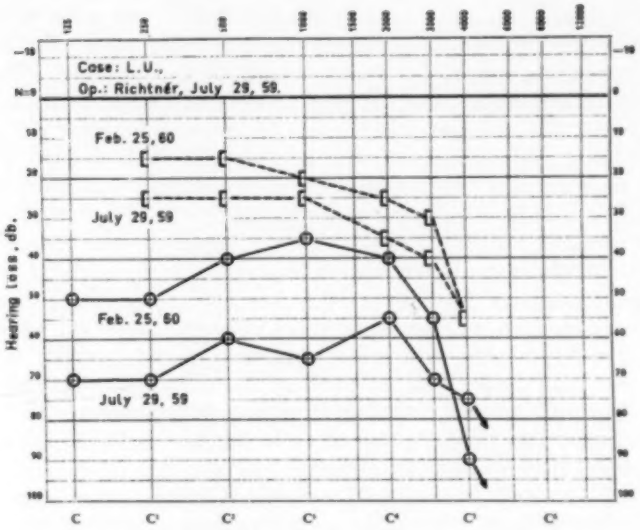

Fig. 5.

At operation it is generally found that a part of the limbus is still present. This should be treated with the greatest care as it provides an excellent bed for the transplant, which is obtained retro-auricularly in all cases in conjunction with the application of the retro-auricular incision. The residues of the drum and of the inner part of the external auditory canal are meticulously denuded of epithelium. The transplant is suspending on the inner part of the external auditory canal and the limbus. If none of the limbus remains a little shelf is

burred in the wall of the auditory canal to provide a seat for the transplant. It is important that there be an open communication between the hypotympanum, the tube and the round window. The transplant is subsequently held in position with Oxycel which is treated with chloromycetin succinate, hyaluronidase and prednisolone. As a rule, this is absorbed within a week or ten days. The oval window is exposed with or without the stapes, another full-thickness skin graft is applied over the defect remaining in the mastoid process and the upper part of the middle ear. In some cases I have left the malleus and incus intact in order to see whether it results in additional improvement of the hearing. The malleus and incus would serve as a sound moderator or Schallschutz. The cases selected for this technique comprise chronic adhesive otitis media, chronic otitis media with or without attic or antrum fistula and with cholesteatoma. In addition there are cases of chronic otitis media with attic or antrum perforation without cholesteatoma. There is also chronic otitis with central perforation and chronic mastoiditis, in some cases with dry ears and in some with suppurating ears. The series also includes some cases of earlier radically operated chronic otitis with continued discharge and greatly impaired hearing as well as cases of clean radically operated cavities with greatly impaired hearing. Altogether my series comprises 200 cases, about 80 of which are included in the series of *cavum minor* technique.

In some cases where the hypotympanum is very low it may prove very difficult to have a graft not healing directly to the mucous membrane of the middle ear; therefore, in some cases I try to place oxycel treated with chloromycetin succinate, hyaluronidase and prednisolone there. Recently I have tried placing a polyethylene tube from the Eustachian orifice over hypotympanum down to the round window and covering it with a full-thickness skin graft. I like to try the polyethylene tube there, because in many cases of otosclerosis treated in this manner very little reaction occurs around a polyethylene tube.

The first follow-up results after operation may be seen in

Table I, which shows the percentage of improvement and impairment and the cases showing no change in the material of 37 cases of chronic otitis operated on with the *cavum minor* technique. This Table shows that one to four months after the operation 49 per cent of the cases showed improvement with a mean gain of 22 db. From five to nine months after the operation 53 per cent showed improvement with a mean gain of 25 db.

TABLE I.

Auditory Results in All Cases After Tympanoplasty *(Cavum Minor)*, 39 Cases.

	1 - 4 Months Post-op.			5 - 9 Months Post-op.			12-18 Months Post-op.		
	No.	Per Cent	M.db	No.	Per Cent	M.db	No.	Per Cent	M.db
Improved	18	49	22	16	48	25	9	53	25
Status quo	18	49	—	17	52	—	7	41	—
Impaired	1	2	13	0	0	—	1	6	19
Total	37	100	—	33	100	—	17	100	—

M.db = mean decibels.

The patients subjected to operation had extremely pronounced auditory impairment throughout. The mean preoperative level of perception for 500, 1,000 and 2,000 cps was 55 db in the patients examined.

Table II shows the auditory results in 72 cases after *cavum minor* tympanoplasty. One month after the operation 38.2 per cent had improved with a mean gain of 20.8 db. The mean preoperative acuity in this series of 55 cases was 60 db. No change was noted in 52.7 per cent. Three months after operation we have 43.2 per cent showing improvement with a mean gain of 22 db. The mean perception in these 53 cases was 62 db. There was no change in 46.2 per cent and 11.6 per cent showed deterioration. Improvement corresponds to a gain of 10 or more than 10 db. Deterioration corresponds to a loss of more than 10 db. Six months after the operation 49 per cent showed improvement in the series of 53 cases, with a mean gain of 21 db; 45.4 per cent showed no change. One year after the operation 51 per cent showed improvement, with gain of 25 db.

In this series 53 cases were examined, with a mean per-

TABLE II.

Results in Tympanoplasty Operations *(Cavum Minor)*.
(Free full-thickness skin graft.)

Time After Op.:	One Month			Three Months			Six Months			One Year			Two Years			Three Years			Four Years		
	No.	%	M.db	No.	%	M.db	No.	%	M.db	No.	%	M.db	No.	%	M.db	No.	%	M.db	No.	%	M.db
Improved	21	38.2	20.8	23	43.2	22	26	49	21	27	51	25	19	50	26	9	39	22	6	—	24
No Change	29	52.7	—	25	46.2	—	24	45.4	—	17	32	—	18	47.4	—	13	55	—	4	—	—
Impaired	5	9.1	—	5	11.6	—	3	5.6	—	9	17	—	1	3.6	—	1	6	—	0	—	—
Total No.	55			53			53			53			38			23			10		
Mean db before Op.			60			62			60			60			61			53			62

Number of cases operated = 72. Mean hearing loss for 500, 1,000 and 2,000 cps in the whole material before operation = 60 db.

ception of 60 db. Two years after the operation 50 per cent were improved and 47.4 remained unchanged, 3.6 had deteriorated. The mean gain was 26 db and the mean perception before operation was 61 db. Three years after the operation we have 39 per cent showing improvement and a mean gain of 22 db. The level before the operation was 53 db. Four years after the operation I have examined only ten cases thus far. This series is too small to justify discussion of the percentage, but six of these ten showed improvement with a mean gain of 24 db, none showed deterioration and four status quo.

It is very interesting to note that three years after the operation the percentage showing improvement drops. I do not know the significance of this decline. Possibly the series is too small to justify conclusions as yet, but after one year I think it will be easier to discuss. In the next series, however, four years after the operation about 60 per cent showed better. This series is also too small to permit definite conclusions.

I personally have treated all these cases postoperatively and I hope to follow them each year now. It would be of great interest to see these patients after two, three or four years. It is not ideal to obtain only 50 per cent improvement, but I hope that with the better technique and the wider experience we gain, our results will eventually become much better.

Recently we have been using more and more fenestration of the oval window rather than fenestration of the horizontal semicircular canal in cases of tympanosclerosis or Paukenscleros (German nomenclature). I have used the Shea method with a vein graft and a polyethylene tube as the stapes in some cases. The results of one case are shown in Fig. 5. Most recently I have used gelfoam instead of a vein graft over the oval window. In these cases and with this technique, there has been no impairment and no dead labyrinth, but the series includes only about ten cases and as yet I can tell you nothing definite about the results of this type of operation. It seems to me to be a good solution in some cases, however, but one must be very sure no acute infection is present.

DR. FURSTENBERG: I am certain that every member of this audience is impressed with the wealth of information on Tympanoplasty that has been presented by our three panelists in this brief period. I have had a chance to study these papers, and I doubt that any important phase of the subject has been overlooked. It would be superfluous reiteration, therefore, if I took your time to dwell in detail upon the many aspects of these presentations so thoroughly and effectively described and explained by the panelists.

In discharging my obligations to this audience I should like to call your attention to Dr. Schuknecht's frank statement that his experience in tympanoplasty now consists of 300 operations performed during the past three years. His failures have been 30 per cent, but some of these have been revised with a successful result. He makes a cogent statement when he warns that, "it is increasingly apparent that it is not always possible to arrest the suppurative process and to reconstruct the middle ear at the same operation." He goes on to emphasize that, "it is also obvious that some failures occur because the nature of the tympanic pathology precluded successful reconstruction."

Dr. Schuknecht lists the contraindications to tympanoplasty namely, a malfunctioning Eustachian tube which fails to ventilate the middle ear adequately. Second, deficient middle ear mucosa. He states that one cannot expect a good functional result "if there is any destruction of mucosa in the area between the Eustachian tube and the round window." Third, hyalinized collagen deposits (tympanosclerosis) in the mucosa of the tympanic cavity. The ossicles may be immobilized by this pathological change, and he emphasizes that "these findings are particularly significant in the presence of a large bone air gap which indicates ankylosis of the ossicles."

Dr. Schuknecht calls upon his vast experience to tell you why tympanic grafts fail. He presents this subject in a detailed manner emphasizing that grafts may fail immediately or many months after the operation. The causes are necrosis due to improper packing, trauma, inadequate blood supply and infection.

Late graft failures may be the result of circumscribed cellulitis or epithelial inclusion cysts. He believes that the majority of these cysts probably arise from epithelial appendages (hair follicles and glands). Puncturing, crushing or traumatizing the graft in a manner which will bury epidermis into its deeper layers may likewise be responsible for inclusion cysts in some instances.

When Dr. Schuknecht makes the statement, "one of the most common causes of tympanoplasty failure is loss of graft substance," I don't believe that he means to imply specifically that this is the precise reason why he did not obtain a good functional result in some of his ears. I feel certain that the influences which were brought to bear in the destruction of the graft also had their serious effect upon other parts of the sound conducting mechanism, scar tissue occlusion of the round window, necrosis or ankylosis of the ossicular chain and hyalinized collagenous material in the mucosa of the tympanum. I have seen many ears with large tympanic perforations in which the auditory function was surprisingly good. On many occasions I have been amazed to discover that the patient with a large perforation in his tympanic membrane had socially adequate hearing in the affected ear. I dwell upon this observation, which I am certain all of you have made many times, because I think we need a new concentration of thought on the causes of conductive deafness in patients who are considered candidates for tympanoplasty. In the vast majority of cases we are dealing with multiple etiological factors and this calls for some serious questioning on our part of what we are doing and why we are doing it.

I always accept the position of moderator with a feeling of embarrassment and even uncertainty as to my ability to do justice to the occasion. I know that it is my responsibility not only to tell you what the panelist's presentation is about but what is more important to tell you what I think of the quality of his discussion. This challenge I accept with pleasure in reviewing Dr. Sullivan's presentation.

He proclaims our indebtedness to Dr. Julius Lempert for the one-stage fenestration operation. He emphasizes the fact

that this magnificent contribution to otology has given not only serviceable hearing to many of the victims of otosclerosis but that it has also inspired a deeper interest in anatomy of the temporal bone and brought together some sincere and intelligent persons from the ranks of the medical sciences to study diseases of the temporal bone and find solutions for their cure. From this operation many observations have been made from which theories have been derived. By careful experimentation some of these theories have been reduced to facts, and more will undoubtedly come in the years ahead.

Dr. Sullivan points out that the aims of the treatment of the chronically discharging middle ear are: *1.* To eliminate the danger of intracranial complications. *2.* To obtain a dry ear. *3.* To preserve and, if possible, to improve the hearing. He speaks with conviction when he states that, "chronic otitis media causes pathological and functional defects of varying degrees, hence the method to restore the hearing must vary." He quotes Stacke who in 1897 said, "No one will bind himself to one method in operating; rather will every thoughtful operator individualize and will modify the method from case to case according to the particular pathological condition." Dr. Sullivan asks, "What do we mean by the terminology, Tympanoplasty?". Well, says he, "It does not mean what it is supposed to mean, namely, a combination of surgery for irradication of chronic ear inflammation and surgical reconstruction of the sound conducting and the sound protecting mechanism. Rarely can it accomplish both of these requisites at one operation" he emphatically states. I agree with him wholeheartedly in this assertion.

Commenting further on tympanoplasty, Dr. Sullivan states, "the results of many, many cases do not justify this tedious, meticulous surgery, because the hearing improvement is not predictable. It is true that the ears can be tested to determine the cochlear function, but you cannot test in an accurate manner how the middle ear will function after the procedure is completed." His knowledge and judgment gained from 30 years of surgical experience in the temporal bone are, I believe, clearly revealed by two statements in the closing para-

graphs of his paper. He says, "I am for any kind of meticulous surgery which cleans the disease from the most remote areas heretofore neglected. As one who has used the dissecting microscope, not just a loop, from my early work in the facial nerve beginning nearly 30 years ago until the present day, I am very cognizant of this fundamental pre-requisite." He leaves little room for argument when he again calls upon his wide experience to say, "Reconstructive aural surgery as a term may suggest a certain uniformity in the operation; it is not, it is different in every case, not only in details but even in the main lines."

We are deeply indebted to Dr. Nils G. Richtner, our guest from Stockholm, for his noteworthy contribution to this panel discussion. He has described a type of tympanoplasty that is called cavum minor.

He has also discussed in considerable detail the difference between free full thickness skin graft and the pedunculated skin and muscle graft. He believes that the latter will give better healing of the graft over the middle ear and make the cavity in the mastoid bone smaller.

He seeks to obliterate the mastoid cavity with a combined skin-muscle flap in order to obtain a small external auditory meatus. He hastens to state, however, that if there is a suppurating otitis he prefers the radical mastoidectomy or some modification of it, and weeks later when the infection has resolved the tympanoplasty is performed.

He describes some ingenious ossicular manipulations in making contact between the incus and the stapes, or malleus and the stapes when there is interruption of the ossicular chain. I had a chance to read his paper hurriedly last night, but much of it pertained to the slides which have just been presented to us.

In closing my comments, permit me to make two more points: first, creativeness is the greatest thing in life. What a thrill it is to discover some surgical procedure that will change the failures of the past into the successes of the future; but we must remember that the consequences of our creations

are the legacy that we leave to the men and women who follow in our footsteps. I cringe from the memories of the fads and fancies of otolaryngology during the period from 1910 to 1930. Eye complications arising from diseases of the nasal accessory sinuses, particularly iritis, choroiditis, and retro-bulbar optic neuritis were over emphasized, and the sinuses suffered radical surgical abuse. The frequent occurrence of sinusitis in infants and children was stressed dogmatically, and the nasal accessory sinuses came in for another surgical assault. We were led astray by the presumption that asthma was the result of a nasal reflex and that radical operations upon the nasal accessory sinuses with removal of their lining mucous membranes were curative measures. Acute mastoiditis which often proved to be a phantom variety was accused of being responsible for diarrhea in infants and children and posted for mastoidectomy on the surgical schedules of many a famous otologist in the years to which I refer. Convictions of the existence of a spheno-palatine ganglion syndrome were fleeting and the administration of vitamins for the restoration of auditory function in perceptive deafness has not been substantiated by scientific studies of an acceptable character. These were some of the whimsical creations of our profession in the period of which I speak. Most of you in this audience today are too young to appreciate the otolaryngological trends of this era, but I think you should know that you were left a legacy from which all of us are still licking our wounds, particularly in the field of medical education.

Finally, I congratulate the panelists for the noteworthy contributions they have made to this important discussion. They have fulfilled their obligation as our essayists by speaking out convincingly and critically of their observations and achievements in this important field of rehabilitation. With freedom of thought and expression extended them as invited panelists, they have taken their responsibilities seriously and provided us with a wealth of information and practical recommendations. This pooling of ideas or theories on the problems of tympanoplasty may lead to final proof or disproof or even to some other solution of our problems.

I doubt that there is any science in which the offering of

theories is taboo. If you have one, let us have it, is my urgent appeal to the members of our profession. Darwin formed a theory about the origin of species. Some of his ideas had to be modified or discarded, but Darwin has not lost stature nor do biologists regard his work as valueless. Incorrect theories about the universe have been offered in the past, but where would astronomy be now if past investigators had ever ceased to form new theories. Theories led to the discovery of some comets and their orbits. Ptolemy presented some astoundingly erroneous theories, but the epochs he created in the progress of astronomy have endured for centuries. The sides of the road up which civilization has travelled are littered with discarded theories, but civilization would have been greatly retarded if inquisitive people had never had the courage to try or to propose anything new.

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Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Arthur V. Hays.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 12-13, 1961.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairman in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Petrarca 332-1, Mexico 5, D. F.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Ohrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. George C. Saunders, 644 Medical Arts Bldg., Portland 5, Ore.
Secretary-Treasurer: Dr. Donald C. Mettler, 1216 S. W. Yamhill St., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Aero Club, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. E. H. Campbell, 133 So. 36th St., Philadelphia 4, Pa.
Secretary-Treasurer: Dr. Raymond Jordan, 121 University Place, Pittsburgh, Pa.
Meeting: Palmer House, Chicago, Ill., October, 1960.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. John F. Tolan, 1118 - 9th Ave., Seattle 5, Wash.
Secretary-Treasurer: Dr. Homer E. Smith, 686 Twelfth Ave., Salt Lake City, Utah.
Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 No. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place:

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. John J. O'Keefe.
Vice-President: Dr. Joseph P. Atkins.
Secretary: Dr. William A. Lell.
Executive Committee: Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, Dr. William A. Lell, Dr. William J. Hitschler, and Dr. Chevalier L. Jackson.

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Cesar F. Villafuerte.
Vice-President: Dr. Napoleon C. Ejercito.
Secretary-Treasurer: Dr. Eusebio E. Llamas.
Directors: Dr. Antonio L. Roxas and Dr. Armando T. Chiong.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Vice-President: Dr. Carson S. Demling, 513 Jenkins Bldg., Pittsburgh 22, Pa.
Secretary-Treasurer: Dr. Clyde B. Lamp, 8101 Jenkins Arcade, Pittsburgh 22, Pa.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

Meeting: University of Vienna, August 29 to September 1, 1960.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Morris E. Krucoff.
Vice-Chairman: Dr. Max J. Fischer.
Secretary: Dr. Adrian J. Delaney.
Treasurer: Dr. Robert D. Ralph.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: Dr. F. T. Land, 13 Newton Place, Glasgow, C. 3.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow, C. 3.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Monterrey 47-201
Mexico 7, D. F.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela.
Treasurer: Dr. Benito Madariaga.
First Vocal: Dr. Rafael González.
Second Vocal: Dr. Juan Oberhauser.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael Garcia Tapia.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DEL NORESTE.

Presidente: Dr. Livio M. Lataza C.
Secretario: Dr. Ramón Prieto.
Tesorero: Dr. José Gomez Galizia.
Vocales: Dres. Enrique del Buono y O. Benjamin Serrano.

SOCIEDAD VENEZOLANA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Gabriel Briceño Romero.
Vice-Presidente: Dr. Silvestre Rincón Fuenmayor.
Secretario General: Dr. Oscar Bustamante Miranda.
Tesorero: Dr. Arturo Marrero Gómez.
Vocales: Dr. Miguel Octavio Russa, Dr. Benjamin Briceño, Dr. Oscar Gonzalez Castillo.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DO RIO GRANDE DO SUL.

President: Dr. Ivo Adolpho Kuhl.
Secretary: Dr. Decio Lisboa Castro.
Treasurer: Dr. Jorge Valentin.

SOCIEDAD PANAMENA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Manuel Preciado.
First Vice-Presidente: Dr. Alonso Roy.
Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. María Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Capt. Maurice Schiff, MC, USN, U. S. Naval Hospital, Oakland, Calif.
Secretary-Treasurer: Lt. Col. James E. Lett, USAF, MC, 209 Tamworth, San Antonio 13, Tex.
Meeting: Concurrent with the Academy of Ophthalmology and Otolaryngology, Chicago, Ill., October, 1960.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. L. D. Lide, Florence, S. C.
President-Elect: Dr. Harry Ross, Anderson, S. C.
Vice-President: Dr. David Stack, Spartanburg, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. C.
Meeting: Jointly with the North Carolina Eye, Ear, Nose and Throat Society in Greenboro, N. C. Headquarters will be in the King Cotton Hotel, Sept. 13-15, 1960.

SOUTH WESTERN LARYNGOLOGICAL ASSOCIATION.
(United Kingdom)

President: Mr. Michael Robert Sheridan, F.R.C.S., 7 Strangways Terrace, Truro, Cornwall.
Secretary and Treasurer: Mr. James Freeman, F.R.C.S., 22 Downs Park West, Bristol, 6.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. George M. Haik, Professor and Head of the Department of Ophthalmology at Louisiana State University School of Medicine, 812 Maison Blanche Building, New Orleans 16, La.
Chairman-Elect: Dr. Mercer G. Lynch, Assistant Professor of Otolaryngology, Tulane University School of Medicine, 3503 Prytania St., New Orleans 15, La.
Vice-Chairman: Dr. Bernard J. McMahon, Director of the Department and Clinical Professor of Otolaryngology, St. Louis University School of Medicine, 8230 Forsythe Blvd., Clayton 24, Mo.
Secretary: Dr. Albert C. Esposito, First Huntington National Bank Building, Huntington, W. Va. Formerly instructor of Ophthalmology, Ohio State University College of Medicine, Columbus, O.
Meeting: St. Louis, Mo., October 31, 1960, to November 3, 1960.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Va.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Va.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Nime K. Joseph, Wheeling, W. Va.
President-Elect: Dr. John A. B. Holt, Charleston, W. Va.
Vice-President: Dr. William K. Marple, Huntington, W. Va.
Secretary-Treasurer: Dr. Albert C. Esposito, Huntington, W. Va.
Director for Two Years: Dr. James T. Spencer, Charleston, W. Va.